THE PLANT DISEASE REPORTER

Issued By

PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

LIBRARY
CURRENT SERIAL RECORD
★ JUL 29 1957 ★
U. S. DEPARTMENT OF AGRICULTURE

Volume 41 Number 7

July 15, 1957

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

THE PLANT DISEASE REPORTER

Issued by

PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Crops Research Division, Plant Industry Station, Beltsville, Maryland
Volume 41, July 15, 1957, Number 7

IN THIS ISSUE

P. B. MARSH reports an investigation of the antidoting effect of some non-toxic salts toward inhibition by copper sulfate of spore germination in Stemphylium sp., page 553.

In greenhouse tests, hexachlorobenzene applied as a seed treatment gave good control of wheat flag smut, according to results reported by LAURENCE H. PURDY, page 558.

RICARDO A. RODRÍGUEZ, CARLOS A. BIANCHINI, and CARLOS A. SOTO describe the "derrite" or "melting-back" disease of coffee, which has been spreading in Costa Rica, and report results of studies on its control, page 560.

Ammonia pellets do not give satisfactory control of fungal decay in packaged cranberries, according to tests reported by PAUL R. HARDING, Jr., page 564.

B. A. FRIEDMAN and M. J. CEPONIS report diseases found in lots of pears shipped from the Pacific Northwest to New York City, page 567.

C. G. WOODBRIDGE, E. C. BLODGETT, and T. O. DIENER describe two types of pear decline, a trouble of unknown cause that has become increasingly serious in the Pacific Northwest, page 569.

A screenhouse provides highly satisfactory conditions for the production of virus-free foundation strawberry stocks, according to J. BERT SMITH and GEO. M. DARROW, page 573.

The antibiotic Cytovirin was effective against several plant viruses in tests reported by REED A. GRAY, page 576.

K. W. KREITLOW, HELEN C. BOYD, D. W. CHAMBERLAIN, and JOHN M. DUNLEAVY have compiled a bibliography of viruses infecting soybean, page 579.

A. M. FINLEY reports that wheat streak mosaic has become an important disease of sweet corn in southwestern Idaho, page 589.

A. L. HOOKER compares various techniques for inoculating oat plants with Septoria avenae and describes methods of measuring varietal reaction to infection, page 592.

A. S. WILLIAMS and L. H. TAYLOR report occurrence of Rathay's bacterial disease of orchard grass in Virginia, page 598.

Occurrence of the nematode Radopholus gracilis in a distinctly drier environment than it is usually associated with is reported by DON C. NORTON, page 599.

J. A. WEST recommends some changes in recovery techniques for the burrowing nematode, page 600.

Reproduction and increase of Meloidodera floridensis in slash pine seedlings apparently does not injure the host, according to pathogenicity tests reported by B. G. CHITWOOD and R. P. ESSER, page 603.

Heavy irrigation increased root-knot injury to tomato seedlings, whereas size of seedlings had no effect, according to results of experiments in Egypt reported by BAKIR A. OTEIFA and DAWOOD M. EL-GINDI, page 605.

N. S. WRIGHT reports that blue spotting of potato tubers, recently identified in British Columbia, occurs only in tubers predisposed by some adverse environmental factor to this type of reaction to handling injury, page 608.

JACK R. WALLIN reports results of pathogenicity studies with monozoospore lines of the potato late blight fungus and describes a technique for isolating single zoospores, page 612.

R. A. HYRE and J. D. WILSON analyze rainfall, temperature, and late blight occurrence records for Ohio, as a basis for forecasting occurrence of the disease in that area, page 616.

N. N. WINSTEAD, ROBERT AYCOCK, and J. M. JENKINS, Jr., report reactions of some 300 watermelon plant introductions and some additional varieties and breeding lines to downy mildew, page 620.

GLENN GARDNER HAHN reports results of American investigations on the life cycle and control of the Phacidiopycnis (Phomopsis) disease of conifers, page 623.

Spread of the Dutch elm disease in North America during 1956 is reported by FRANCIS W. HOLMES, page 634.

IN THIS ISSUE (Continued on page 651).

Vol. 41, No. 7--PLANT DISEASE REPORTER--July 15, 1957

AN EFFECT OF SALTS IN ANTIDOTING THE INHIBITION OF GERMINATION OF SPORES OF STEMPHYLIUM SP. BY COPPER SULFATE

P. B. Marsh[1]

Abstract

An important part of the current theory of the mechanism of toxic action of copper on fungus spores (1) is based on data relating to an antidoting effect of various non-toxic salts toward inhibition of spore germination by copper sulfate (2) and on the influence of such salts in decreasing copper absorption by the spores (3). Since reports demonstrating the copper-antidoting phenomenon have been limited to only a very few fungi, it was considered worthwhile to report here its occurrence with an additional fungus, a Stemphylium.

With calcium chloride or magnesium sulfate, a molarity of about 60 to 75 times that of the copper sulfate was required to provide a strong antidoting effect as measured in terms of spore germination, whereas with potassium chloride or sodium chloride the required ratio was of the order of 3000. This higher antidoting effectiveness of the divalent-cation salts in comparison with the monovalent-cation salts is in general accord with previous data and theories (1, 2, and 3).

BACKGROUND

Lin (2) demonstrated several years ago that when spores of the fungus Monilinia fructicola were suspended in a solution containing copper sulfate the germination-inhibiting action of the copper sulfate could be antidoted by any of several non-toxic salts added to the solution. Magnesium sulfate, calcium sulfate, magnesium chloride, magnesium nitrate, calcium chloride, calcium nitrate, potassium sulfate, potassium nitrate, potassium chloride, sodium chloride, and various other salts exhibited this action. The concentration of the antidoting salt required to antagonize the effect of the copper was related to the valence of the cation, with magnesium and calcium salts being effective at lower concentrations than potassium or sodium salts. The present author (3), working with the same fungus, showed that the copper-antidoting effect was correlated with a large decrease in copper absorption in the presence of the antidoting salts, and that the divalent cations were again more effective than the monovalent ones.

METHODS

The fungus used was a Stemphylium designated in this laboratory under the number 1203.1, originally isolated from raw cotton from Knoxville, Tennessee, in 1937 and identified by Miss Vera Charles in 1940. It is listed at the American Type Culture Collection by the number 9569. Spores were produced by growing the fungus on a circle of filter paper supported by glass beads at the surface of a mineral-salt solution in a 250-cc Erlenmeyer flask. The mineral-salt solution contained NH_4NO_3, KH_2PO_4, K_2HPO_4, and $MgSO_4 \cdot 7H_2O$ at concentrations of 1.0, 0.9, 0.7, and 0.75 grams per liter respectively. At the end of 2 weeks' incubation at approximately 20° C, the cultures were shaken to dislodge the spores. The spores were then washed by centrifuging three times in 40-cc portions of glass-distilled water to remove salts carried over from the growth medium.

Germination tests were carried out in 10-cm, flat-bottomed, Pyrex petri dishes, using in each case a total of 10 cc of liquid medium, made up with glass-distilled water. In each case 0.01% glucose was present in the medium. Germination counts were made after 24 hours at room temperature. Each percentage germination figure reported is an average from counts in each of five areas in the dish, each area involving at least 100 spores.

[1] Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

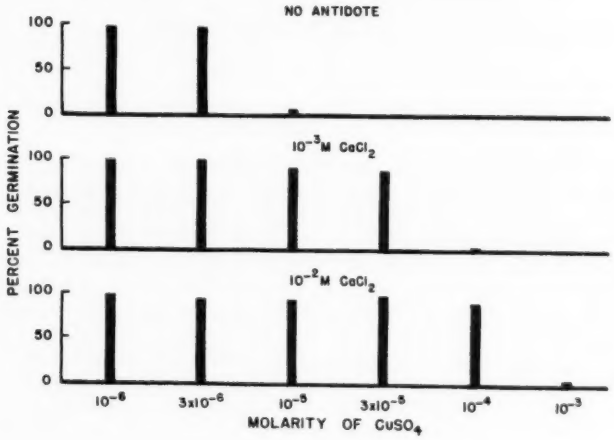


FIGURE 1. Percent germination of spores of Stemphylium 1203.1 in the presence of copper sulfate alone and in the presence of copper sulfate plus calcium chloride at two different concentrations.

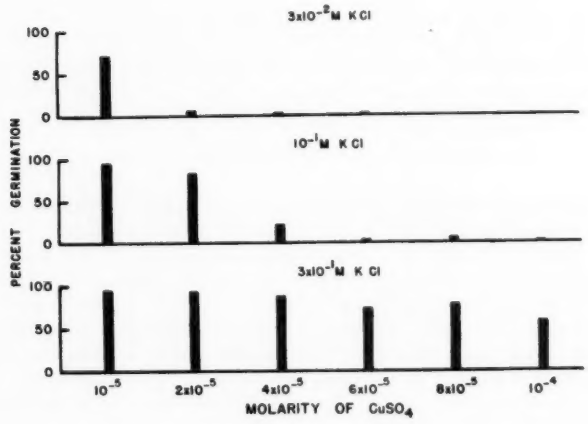


FIGURE 2. Percent germination of spores of Stemphylium 1203.1 in the presence of copper sulfate plus potassium chloride at three different concentrations.

Table 1. Percent germination of spores of Stemphylium 1203.1 in the presence of various binary combinations of copper sulfate and calcium chloride. Data demonstrate antidoting effect of calcium chloride toward toxic action of copper sulfate.

Copper Sulfate Concentration:	Calcium Chloride Concentration:				
	10^{-4}M	$3x10^{-4}$M	10^{-3}M	$3x10^{-3}$M	10^{-2}M
10^{-5}M	47	55	85	92	96
$2x10^{-5}$M	30	38	79	90	96
$4x10^{-5}$M	8	30	88	95	100
$6x10^{-5}$M	0	0	0	12	100
$8x10^{-5}$M	0	0	0	5	96
10^{-4}M	0	0	0	5	92

Table 2. Percent germination of spores of Stemphylium 1203.1 in the presence of $5x10^{-5}$M copper sulfate and an antidoting salt. Data demonstrate differences in antidoting effectiveness of the four salts.

Antidoting Salt	Antidoting salt at concentration of:							
	10^{-4}M	$3x10^{-4}$M	10^{-3}M	$3x10^{-3}$M	10^{-2}M	$3x10^{-2}$M	10^{-1}M	$3x10^{-1}$M
$CaCl_2$	19	31	27	88	94	--	--	—
$MgSO_4$	0	0	0	88	81	--	--	--
KCl	0	0	0	0	0	2	53	89
NaCl	0	0	0	0	1	2	30	86

EXPERIMENTAL

In a first experiment (Fig. 1) spore germination was determined in a range of concentrations of copper sulfate from 10^{-6}M to 10^{-3}M under three conditions: (a) in the absence of any added salt, (b) in the presence of 10^{-3}M $CaCl_2$, and (c) in the presence of 10^{-2}M $CaCl_2$. At the two lowest concentrations of copper sulfate a very high percentage of the spores germinated in all cases, but at copper sulfate concentrations in the range of 10^{-5}M to 10^{-4}M there was little or no germination in the no-salt plates and high germination in the plates containing $CaCl_2$ in addition to the copper sulfate. At the 10^{-4}M concentration of $CuSO_4$ it was seen that a 100-fold ratio of $CaCl_2$ to $CuSO_4$ (10^{-2}M/10^{-4}M) was adequate to provide good germination, while a 10-fold ratio (10^{-3}M/10^{-4}M) was not adequate.

In a second experiment (Fig. 2) KCl was tested as an antidoting salt at three concentrations in antagonism to the toxic effect of copper sulfate in the range of 10^{-5}M to 10^{-4}M. Again a definite antidoting effect was noted. It had been expected from previous work with Monilinia (2, 3) that KCl would be required at higher ratios in proportion to the copper sulfate than $CaCl_2$ in order to cause good antidoting, and this was in fact found to be the case. It may be noted that at concentrations of $CuSO_4$ of 2-, 4-, 6-, and $8x10^{-5}$M, a major degree of antidoting was provided by KCl at KCl/$CuSO_4$ ratios of 5000, 7500, 5000, and 3750 respectively, but not at ratios of 1500, 2500, 1666, and 1250; these data suggest that the ratio required for strong antidoting (80 percent germination or better) is probably slightly above 3000. This estimate is further borne out by the observation that the antidoting ratio at 10^{-4}M $CuSO_4$ is 3000 ($3x10^{-1}$M/10^{-4}M) and that this ratio allowed about 55 percent germination.

In a third experiment (Table 1) the antidoting action of $CaCl_2$ toward $CuSO_4$ was investigated with a greater number of concentration combinations. At $CuSO_4$ concentrations of 1-, 2-, 4-, 6-, and $8x10^{-5}$M and 10^{-4}M, 80 percent germination or higher was provided at $CaCl_2$/$CuSO_4$ ratios of 100, 150, 25, 166, 125, and 100, but not at ratios of 30, 50, 7, 50, 37, and 30. If one admits the possibility of experimental error in the "25" in the former series, it seems that the ratio of $CaCl_2$/$CuSO_4$ required for strong antidoting is in the general range of 75. While this figure is obviously only a rough estimate, it is in general accord with the fact that a 100-fold ratio was adequate and a 10-fold ratio inadequate in the $CaCl_2$ experiment of Figure 1. The estimated ratio of 75 for $CaCl_2$ is also clearly much lower than the approximately 3000 estimate for KCl.

In a fourth experiment (Table 2) two additional salts were tested in respect to their possible antidoting potentialities, namely $MgSO_4$ and NaCl. In this experiment, it is seen that $MgSO_4$ behaved rather similarly to $CaCl_2$, both providing good antidoting at a salt/$CuSO_4$ ratio of 60 ($3x10^{-3}$M concentration of either salt in comparison with $5x10^{-5}$M $CuSO_4$). The NaCl was similar in effectiveness to the KCl, both providing good antidoting at a ratio of 6000 ($3x10^{-1}$M salt) and moderate antidoting at a ratio of 2000 (10^{-1}M salt). These data are then in general accord with the data of Figure 2, from which a figure of 3000 was estimated as the required ratio for good antidoting (80 percent germination) with KCl.

In reviewing the data of the four experiments of Figures 1 and 2 and Tables 1 and 2, it seems that a much higher concentration ratio of either KCl or NaCl to $CuSO_4$ is required for good antidoting -- around 3000 -- than is required for either $CaCl_2$ or $MgSO_4$ -- around 60 to 75. In Lin's (2) experiments the corresponding ratio for KCl and NaCl is about 12,500 and for $CaCl_2$ and $MgSO_4$ about 12.5 and 25 respectively. Although the data are not extremely precise in either case, it seems that with the Monilinia the difference in effectiveness between the divalent-cation and monovalent-cation salts was greater than in the experiments reported here with Stemphylium.

The antidoting phenomenon is interpreted from prior evidence with Monilinia to be brought about by a decrease which the antidoting cation causes in the amount of copper absorbed by the spore (3), the effect being considered to be in the nature of a base-exchange type of phenomenon. In the case of silver, Miller and McCallan (4) have shown recently that mercury(I) and mercury (II), and to a lesser degree copper, competitively inhibited the absorption of silver by spores.

Literature Cited

1. HORSFALL, J. G. 1956. Principles of Fungicidal Action. 279 pp. Chronica Botanica Co., Waltham, Mass.
2. LIN, C-K. 1940. Germination of conidia of Sclerotinia fructicola, with special reference to the toxicity of

copper. Cornell University Memoir 233: 1-33.
2. MARSH, P. B. 1945. Salts as antidotes to copper in its toxicity to the conidia of Sclerotinia fructicola. Phytopathology 35: 54-61. 1945.
3. MILLER, L. P., and S. E. A. McCALLAN. 1957. Toxic action of metal ions to fungus spores. Agricultural and Food Chemistry 5: 2, 116-122. February.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND

TOXICITY OF HEXACHLOROBENZENE TO UROCYSTIS AGROPYRI[1]

Laurence H. Purdy[2]

The toxicity of hexachlorobenzene (HCB) is generally regarded to be specific for the wheat bunt fungi (Tilletia spp.). According to Lhoste and Ravault (1) this chemical is not effective against any of several species of fungi belonging to genera within the four main classes of fungi. Observations in our laboratory have shown that the growth of the commonly occurring air-borne saprophytes (species of Penicillium, Aspergillus, and Rhizopus) was not adversely affected by HCB. This apparent specificity for wheat bunt is the main objection to the use of HCB as a seed treatment for wheat because it fails to provide protection against seedling blight or seed decay organisms.

With regard to other smuts than wheat bunt, Lhoste and Ravault (1) have shown that HCB is not effective against Ustilago hordei, U. nuda, and U. nuda tritici. Machacek (2) presented evidence that HCB failed to control loose smut of oats (Ustilago avenae) and covered smut of barley (U. hordei). These reports were confirmed by the results of tests with these same species, as summarized in Table 1. Good control was obtained with Panogen 15 while HCB was ineffective. The evidence seems conclusive, therefore, that the highly toxic action of HCB on species of Tilletia does not extend to species of Ustilago. Little is known of its toxic effects on smut species of other genera. In view of the close taxonomic relationship between Tilletia and Urocystis, tests were made to determine whether HCB is effective against flag smut of wheat (Urocystis agropyri (Preuss) Schroet.) and whether the seed-borne phase of the disease could be controlled by seed treatment with HCB.

Table 1. Percent smut in seed treatment tests with naturally infested oats and barley in 1956.

Treatment	Ounces per bushel	Oats	Barley
Check		14	4
Anticarie (40% HCB)	1	11	7
Panogen 15	3/4	4	0

In seed treatment tests conducted in the greenhouse, seed of the wheat variety Federation was inoculated with flag smut spores. The fungicides were applied as for bunt seed treatment tests (3). Anticarie and Sanocide (both 40% HCB) and a mercurial check, Panogen 15 (2.2% cyano (methylmercury) guanidine) were used. The treated seed was planted in the greenhouse at 55° to 60° F. Smut percentages were based on the number of smutted plants per row. Three replications of each treatment were used and the test was duplicated.

The results of the two tests, expressed as average percentages, are summarized in Table 2. High percentages of smut developed in the untreated check rows of both tests, and good control was provided by both the mercury and HCB preparations. Thus it is apparent that the sphere of activity of HCB is broader than the previously indicated specificity for wheat bunt.

---

[1]Cooperative investigations of the United States Department of Agriculture, Crops Research Division, Agricultural Research Service, and the Agricultural Experiment Stations of Idaho, Oregon, and Washington. Scientific Paper No. 1622, Washington Agricultural Experiment Stations, Pullman, Washington.

[2]Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Pullman, Washington.

Table 2. The effectiveness of hexachlorobenzene seed treatment in the control of flag smut of wheat originating from seed-borne spores.

Treatment	Ounces per bushel	Percent smut 1956	1957
Check		47	21
Hexachlorobenzene[a] (40%)	1	0	3
Panogen 15	3/4	0	1

[a] Anticarie brand was used in 1956 and Sanocide brand was used in 1957.

Literature Cited

1. LHOSTE, J., and L. RAVAULT. 1949. Contribution a l'étude de l'hexachlorobenzene, produit actif contre les spores de Tilletia tritici Bjerk. 2nd Congress of Crop Protection, Londres, 9 pp., 1951.
2. MACHACEK, J. E. 1956. Co-operative seed treatment trials -- 1955. Plant Dis. Reptr. 40: 33-36.
3. PURDY, L. H. 1955. Regional seed treatment tests for the control of seed-borne and soil-borne common smut of winter wheat in the Pacific Northwest. Plant Dis. Reptr. 39: 844-849.

UNITED STATES DEPARTMENT OF AGRICULTURE, CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, AND THE AGRICULTURAL EXPERIMENT STATIONS OF IDAHO, OREGON, AND WASHINGTON

STUDIES ON THE CONTROL OF "DERRITE" DISEASE OF COFFEE CAUSED BY PHYLLOSTICTA COFFEICOLA DEL.

Ricardo A. Rodríguez, Carlos A. Bianchini, and Carlos A. Soto

INTRODUCTION

A new disease has been spreading year after year to new coffee plantations in Costa Rica, and at the present time it is a constant menace to farmers who see their crops greatly diminished by the malady.

It was observed on coffee not so long ago, in the highlands north of Alajuela and Heredia. Now it has moved into the Central Plateau and is found in almost every neighboring upland area cultivated with coffee in this country.

Little work is being done on this subject, and no literature on the control of the disease is available.

The disease is characterized by blighting of the new shoots and leaves and, as a result, growth is delayed or completely stopped in the most severe cases. This characteristic symptom, a melting-back, is described by the Spanish word "derrite", which is used as the popular name for the disease.

According to its present distribution, "derrite" seems to be favored by cool and humid climatic conditions.

SYMPTOMS

Phyllosticta coffeicola Del. attacks coffee from the seedling to the adult stage, apparently being more severe under shade conditions, according to Alvarado (1). Two kinds of symptoms can be set apart based on the plant parts attacked: lesions on the leaves and lesions on the twigs

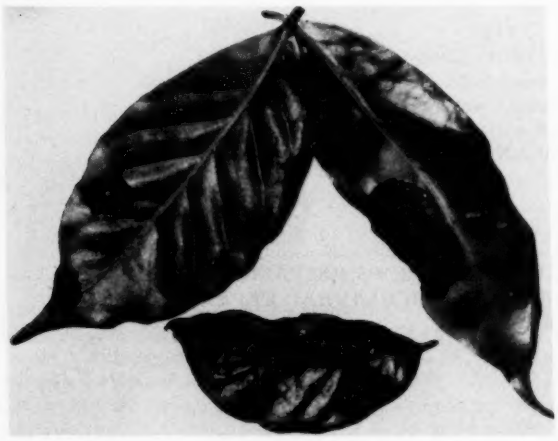


FIGURE 1. Phyllosticta coffeicola Del. on leaves of coffee. Note characteristic curving of midrib of small leaf, resulting from a marginal lesion.

FIGURE 2. Twig blight phase of "derrite" on Coffea arabica, like that caused by Phomopsis sp. on C. liberica according to Castaño (2).

Leaf lesions are commonly apical or on the borders, forming irregular rounded spots with concentric ridges, or almost circular lesions in the leaf lamina that may sometimes lose their centers (Fig. 1). Young lesions are black or very dark brown, but turn rusty and

Table 1. Inhibitory effect of certain fungicides and antibiotics on the development of Phyllosticta coffeicola, measured on treated paper discs.

Treatment	Concentration	Average diameter of inhibition area (mm)
FUNGICIDES	gm per liter	
T1 -- Tuzet (Bayer) Zn dithiocarbamate-TMTD (As 1.6%)	0.9	32.96
T3 -- Orthocide 50 (Captan)	3.2	34.51
T4 -- Fuclasin Ultra (90% Ziram)	2.7	22.93
T6 -- Emmi (mercury compound)	0.3	40.83
T7 -- Phix (phenyl mercury acetate 22%)	0.3	40.46
T8 -- Solbar (70% barium sulfate)	2.0	13.10
T11 -- Oxicob (copper oxychloride)	2.7	0.00
T19 -- Karathane WD (dinitro-phenol)	0.6	0.00
T17 -- Check	---	0.00
T18 -- Manzate (maneb)	2.7	16.85
T19 -- Stauffer N-521	0.4	14.09
ANTIBIOTICS	ppm	
T2 -- Chloromycetin (Parke Davis)	100	0.00
T5 -- Filipin (Upjohn Co.)	100	18.24
T9 -- Acti-dione (Upjohn Co.)	2	0.00
T10 -- Rimocidin (Pfizer)	1,000	40.48
T12 -- Oligomycin (Pfizer)	100	14.51
T13 -- Agri-mycin 100 (Pfizer)	200	0.00
T14 -- Anisomycin (Pfizer)	100	0.00
T15 -- Streptomycin sulfate (Pfizer)	100	0.00

leathery in texture as they get old. From early stages, lesions show on both sides of the leaf lamina with rather definite borders. Tissues adjacent to a marginal lesion are deformed and the leaf is usually curved toward the blighted part. In environments with high moisture conditions, the lesions show a velvet-like mass of sporulating tissues.

Infected leaves that are not shed by the tree constitute a source of inoculum that can eventually be distributed by wind and rain to healthy plants. From field observations Soto believes that insect transmission can be proved.

Attack on the twigs appears like a fire that rapidly kills the apical bud and the first pair of leaves, and stops as it reaches the lignified portion of the branch (Fig. 2). J. J. Castaño

Table 2. Results of field treatment showing differences in the control of Phyllosticta coffeicola with fungicides and antibiotics.

Treatment	Average rating before treatment	Average rating 8 weeks after start of treatment
1% Griseofulvin dust (Merck antibiotic)	3.66	1.00
Tuzet (TMTD - As Zn dithiocarbamate)	4.00	0.5
Phix - (22% Phenyl mercury acetate)	5.18	1.5
Emmi (Mercury compound)	3.33	0.5
Orthocide 50 (Captan)	4.18	0.5
Fuclasin Ultra (Ziram)	4.18	2.0
Tritoflorol (Zineb)	5.00	2.2
50% wettable griseofulvin 100 ppm	3.80	2.2
Actidione, 2 ppm (Upjohn antibiotic)	4.50	1.0
Check	4.22	2.2

(2), believes that twig blight in Coffea liberica is caused by Phomopsis sp., a fungus closely related to Phyllosticta.

LABORATORY TESTS

Using a somewhat modified Schaal and Johnson's method (3) three standard potato-dextrose agar Petri plates were inoculated with a water suspension of mycelial pieces. Three paper discs (12 mm in diameter), were previously treated by immersion for 5 minutes in the antifungal suspension and set in each plate.

The results were interpreted by measuring, in millimeters, the diameter of the inhibition area after 72 hours' incubation at 24° C.

The data, expressed as the average of 9 replicates per treatment, are shown in Table 1.

The present experiment shows that Emmi, Rimocidin and Phix were the most effective fungicides against Phyllosticta coffeicola as tested in the laboratory. Orthocide and Tuzet are next, in decreasing order of effectivity.

Other fungicides and antibiotics included in this experiment did not show promise in the control of the fungus at the concentrations in which they were used.

FIELD EXPERIMENTS

A badly infested plot and 12 adult plants per treatment were chosen for this trial. Before spraying was done, the amount of infection was rated from 0 to 8 according to the following scale:

0 -- Plants free from infection
2 -- 25 percent leaves and shoots blighted
4 -- 50 percent leaves and shoots blighted
6 -- 75 percent leaves and shoots blighted
8 -- All shoots blighted

Sprays were applied four times during October and November at intervals of every 2

weeks. A second rating was obtained after the fourth spray, based on observations on the leaves and twigs newly blighted.

The results shown in Table 2 are expressed as the average of 12 plants or replicates.

A close relationship between laboratory and field experiments can be observed: Emmi, Tuzet, Phix and Orthocide showed similar effects in both experiments. A third rating will be obtained in the next rainy season, since these data were taken during the dry period. More conclusive data will then be available for a final estimate of the results obtained.

The antibiotic Griseofulvin was acquired in time for the field experiments and was included although it was not previously tested in the laboratory.

It was not possible to test Rimocidin and other antibiotics in the field experiment because we had only small laboratory samples.

A more extensive program in the study of these promising fungicides for the control of "derrite" underway.

Literature Cited

1. ALVARADO, J. A. 1935. Tratado de Caficultura Práctica. Vol. 1, Chapter VIII. Guatemala, Centro América.
2. CASTAÑO, J. J. 1954. Una afección nueva en Coffea liberica. Revista Cafetera de Colombia, Centro Nacional de Investigaciones de Café. Chinchiná, Caldas. 12 (126): 4123-4125.
3. SCHAAL, L. A., and G. JOHNSON. 1956. The inhibitory effect of phenolic compounds on the growth of Streptomyces scabies as related to the mechanism of scab resistance. Phytopathology 45: 626.

SECCIÓN DE FITOPATOLOGÍA, MINISTERIO DE AGRICULTURA E INDUSTRIAS, SAN JOSÉ COSTA RICA

PRELIMINARY TESTS OF AMMONIA PELLETS FOR CONTROL OF FUNGAL DECAY OF PACKAGED CRANBERRIES

Paul R. Harding, Jr.

Summary

An experiment was performed to determine the capacity of ammonia for controlling fungal decay in cranberries packaged in polyethylene film. Inoculation consisted of dusting an abundance of conidia of Penicillium funiculosum Thom on the cranberries without injury to their surface. Treatment consisted of including in each treated package one dry pellet capable of releasing ammonia on absorption of moisture. Four different conditions of moisture were used to determine the most effective method of applying this treatment. After 12 days of storage in a forced-air ventilated room having a temperature of 75° F and a relative humidity of 62 percent, percentages of berries with decay and ammonia injury were determined.

Under the conditions described, treatment with pellets that depend on absorption of moisture for release of ammonia is effective against decay only when the cranberries are packaged in nonperforated plastic film. Such unventilated bags are able to maintain a humidity high enough to bring about a release of enough ammonia to give some protection against fungal decay. Because of the injury caused by ammonia in samples with considerable control of decay, it was concluded that the ammonia-pellet treatment as used here is not satisfactory for prevention of decay in cranberries.

INTRODUCTION

A large percentage of Early Black cranberries shipped from Wareham, Massachusetts, to Los Angeles, California, in October 1955 developed fungal decay in ice-refrigerated railway cars in which temperatures ranged from 50° to 65° F. By contrast, cranberries from the same source shipped in mechanically refrigerated railway cars at temperatures near 40° arrived in excellent condition. To determine the nature of the decay, isolations were made from diseased berries and several species of fungi were obtained. Among these were the usual fruit-rotting fungi such as Sporonema oxycocci Shear, Diaporthe vaccinii Shear, Pestalotia vaccinii (Shear) Guba, and Guignardia vaccinii Shear. The most frequently occurring organism, however, was Penicillium funiculosum Thom. Shear et al.[1] listed two unidentified species of Penicillium as commonly causing storage rot in cranberries, but since they gave no descriptions it is uncertain that either of their species was P. funiculosum.

The ammonia pellets used in these tests were developed by Gunther et al.[2] at the Citrus Experiment Station in Riverside, California, for controlling postharvest decay of citrus fruits caused by the blue and green molds (Penicillium digitatum Sacc. and P. italicum Wehmer). That they have been used with some success to prevent postharvest decay in citrus fruits influenced the writer to test them as a possible means of controlling storage rot in cranberries. Each pellet weighs 2 grams and contains 6% ammonia in a formulation composed of soda ash and ammonium sulfate in a carrier known as bentonite. Since the pellets depend on absorption of moisture for release of ammonia, an integral part of the study was to determine the moisture condition at which the treatment is most effective.

METHODS AND MATERIALS

In November 1956, Early Black cranberries free from any sign of injury or decay were selected for packaging at the rate of 30 per 4 x 6-inch polyethylene bag. Inoculation consisted of dusting an abundance of conidia of Penicillium funiculosum on the cranberries without in-

[1] Shear, C. L., N. E. Stevens, and H. F. Bain. 1931. Fungous diseases of the cultivated cranberry. U. S. Dept. Agr. Technical Bulletin No. 258, pp. 1-58.

[2] Gunther, F. A., M. J. Kolbezen, R. C. Blinn, E. A. Staggs, J. H. Barkley, G. B. Wacker, L. J. Klotz, C. N. Roistacher, and A. El-Ani. 1956. Ammonium succinate and inorganic ammonia producing materials as fungicides for the control of blue-green mold decay of citrus fruits. Phytopathology 46: 632-633.

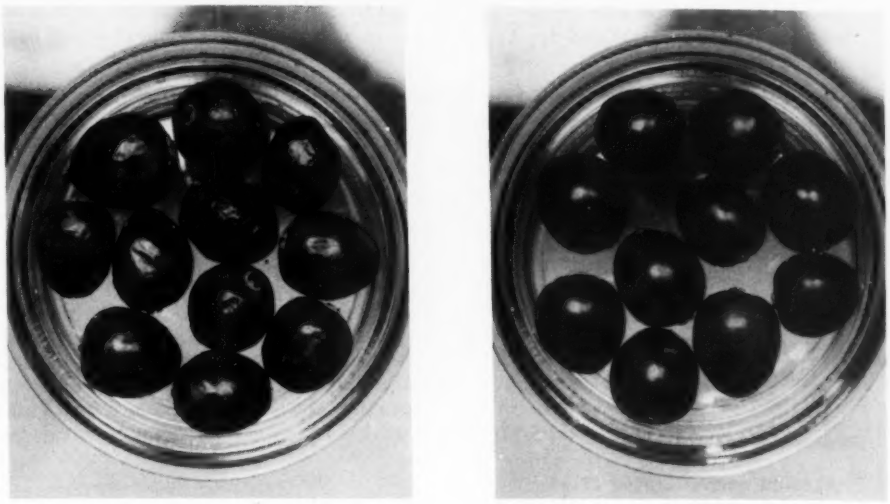


FIGURE 1. A -- Decay pattern of the type caused by Penicillium funiculosum Thom or Guignardia vaccinii Shear. B -- Healthy cranberries.

juring their surfaces. Treatment consisted of including in each treated package one dry 2-gram ammonia pellet. The basic experimental arrangement was as follows: five bags inoculated and treated; five bags inoculated but not treated; five bags not inoculated but treated; and five bags neither inoculated nor treated. This arrangement was used in four series, each with a different condition of moisture as follows: 1) berries wet in nonperforated bags; 2) berries wet in perforated bags; 3) berries dry in nonperforated bags; and 4) berries dry in perforated bags. Each perforated bag had 10 holes of 4-mm diameter. The experiment was run under the four different conditions of moisture to determine the most effective method of applying the ammonia-pellet treatment, the rate of release of ammonia depending on the amount of moisture present in the package. After 12 days in a forced-air ventilated room at a temperature of 75° F and a relative humidity of 62 percent, percentages of the berries decayed and injured by ammonia were determined.

RESULTS AND CONCLUSIONS

Under the first condition of moisture (Table 1), treatment reduced decay in the inoculated berries from 57.3 to 14 percent, while injury due to ammonia was 15.3 percent. In the comparable samples of noninoculated berries treatment reduced decay from 32 to 6.7 percent, while injury due to ammonia was 38 percent. Under the second condition of moisture, treatment caused little or no reduction in decay, and there was no ammonia injury. Under the third condition of moisture (Table 1), treatment reduced decay somewhat, but ammonia injury occurred. Under the second and third conditions, the pellets were dry and showed very little evidence of breakdown at the end of the holding period. Under the fourth condition of moisture (Table 1), treatment did not reduce decay and ammonia injury was not present.

The results indicate that when cranberries are stored in a forced-air ventilated room at a temperature of 75° F and a relative humidity of 62 percent, treatment with pellets that depend on absorption of moisture for release of ammonia is more effective against decay when the berries are packaged in nonperforated plastic film. Nonperforated bags containing dry berries developed an equilibrium relative humidity of 92 percent in about 35 minutes. In contrast, perforated bags containing dry berries kept the same relative humidity as that of the storage room. Only in the nonperforated bags was the humidity high enough to bring about a

Table 1. Effect of inoculation with Penicillium funiculosum and inclusion of dry ammonia pellets on decay of wet and dry cranberries in perforated and nonperforated bags held 12 days at 75° F[a].

Experimental conditions	Percent of berries decayed	Percent of berries injured by ammonia
1. Wet berries in nonperforated film:		
inoculated, treated	14	15.3
inoculated, nontreated	57.3	0
noninoculated, treated	6.7	38
noninoculated, nontreated	32	0
2. Wet berries in perforated film:		
inoculated, treated	28.7	0
inoculated, nontreated	26.7	0
noninoculated, treated	24	0
noninoculated, nontreated	26	0
3. Dry berries in nonperforated film:		
inoculated, treated	26	24.7
inoculated, nontreated	44	0
noninoculated, treated	22.6	16.7
noninoculated, nontreated	36.7	0
4. Dry berries in perforated film:		
inoculated, treated	22.7	0
inoculated, nontreated	22	0
noninoculated, treated	16.7	0
noninoculated, nontreated	18.7	0

[a] Each value is based on 30 berries in each of 5 bags.

release of ammonia adequate to give some protection against fungal decay. However, because of the injury caused by ammonia, it appears that the ammonia-pellet treatment as used in this experiment is not satisfactory as a means of decay prevention in cranberries. Injury due to excessive accumulation of ammonia in nonperforated bags is characterized by dark-blue areas that are usually soft.

Only under the first condition of moisture wherein the cranberries were wet and in nonperforated bags was there definite evidence that applying conidia of Penicillium funiculosum to the surface increased decay.

Isolations from decayed berries showed Penicillium funiculosum to be common in all samples. Other agents of decay common among all samples included Sporonema oxycocci, Diaporthe vaccinii, Pestalotia vaccinii, and Guignardia vaccinii. The decay pattern characterized by whitish soft spots, as shown in Figure 1, is of the type caused by Penicillium funiculosum or Guignardia vaccinii, the two sometimes being mixed in a single lesion.

AGRICULTURAL MARKETING SERVICE, BIOLOGICAL SCIENCES BRANCH, QUALITY MAINTENANCE AND IMPROVEMENT SECTION, POMONA, CALIFORNIA

DISEASES OF D'ANJOU PEARS AT THE NEW YORK CITY MARKET[1]

B. A. Friedman and M. J. Ceponis

During January and February 1957, ripening studies were conducted on 11 lots of D'Anjou pears that had originated from eight different sources in the pear-growing regions around Wenatchee, Washington, and Medford, Oregon. On arrival at the market, decay ranged from 0 to 2.6 percent and averaged 0.7 percent. After ripening for periods from 7 to 16 days at 70° F (R.H. about 85 percent), decay ranged from 0.8 to 4.7 percent and averaged 1.8 percent. The principal rots found were blue mold (Penicillium), gray mold (Botrytis), and bull's-eye (Neofabraea) (Table 1.).

The 4 lots of pears from Washington State had been stored at the shipping point in non-perforated polyethylene liners and, at the arrival examination in New York, nearly 10 percent of the fruit was damaged by a pithy brown core breakdown. This physiological condition was apparently caused by high concentrations of CO_2 within the packages. After the ripening period, almost 18 percent of the pears were found damaged by this internal breakdown (Table 2). The remaining seven lots of pears, all from Oregon, had been previously stored in perforated liners and did not show this breakdown. At arrival they showed a slight amount of pear scald which increased considerably during the ripening period to about 7 percent (Table 3).

Table 1. Types of decay found in test packages of D'Anjou pears at the New York City market.

Lot No.[a]	Number pears examined[b]	Type and percent of rot: Blue mold	Gray mold	Bull's-eye	Cladosporium	Alternaria	Rhizopus
1	731	1.1	1.1	0	0	0	0
2	753	0.5	0.1	0	0	0	0.1
3	618	1.6	0.3	0	0	0	0.2
4	766	1.0	0.1	0	0	0	0
5	481	0.6	0.4	3.1	0.2	0	0
6	532	1.5	0.2	0	0	0	0
7	557	1.4	0.2	0.2	0	0	0
8	500	0.8	0.0	0.2	0	0	0
9	592	1.0	0.2	0.2	0	0.2	0
10	547	0.2	0.2	0.5	0	0	0
11	575	0.9	0.5	0	0	0	0
Total	6652						
Average		1.0	0.3	0.3	<0.1	<0.1	<0.1

[a]Lots 1 to 4 originated from Washington State and lots 5 to 11 from Oregon.
[b]Examinations were made at arrival and during ripening.

[1]One of a series of notes from the Market Pathology Laboratory, Biological Sciences Branch, United States Department of Agriculture at New York, New York, to provide accurate information on market spoilage of fresh fruits and vegetables.

Table 2. Pithy brown core breakdown found in Washington State-grown D'Anjou pears at the New York City market.

Lot no.	Number of pears examined: On arrival	Number of pears examined: During ripening	Percentage of pithy brown core breakdown: On arrival: Slight	On arrival: Moderate	On arrival: Severe	On arrival: Damaged[a]	After ripening: Slight	After ripening: Moderate	After ripening: Severe	After ripening: Damaged[a]
1	45	670	15.6	6.7	2.2	8.9	27.9	12.8	8.1	20.9
2	36	711	25.0	8.3	0	8.3	27.3	10.5	9.4	19.9
3	36	569	8.3	0	0	0	10.5	2.1	0.9	3.0
4	36	721	11.1	5.6	16.7	22.3	27.0	13.7	11.0	24.7
Total	153	2671								
Average			15.0	5.2	4.6	9.8	23.8	10.2	7.7	17.9

[a]Slight breakdown was not considered of commercial significance; moderate and severe breakdown was considered to damage the fruit and to be scorable against grade standards.

Table 3. Incidence of scald found on Oregon-grown D'Anjou pears at the New York City Market.

Lot no.	Number of pears examined: On arrival	Number of pears examined: During ripening	Percentage of scald: On arrival: Slight	On arrival: Moderate	On arrival: Severe	On arrival: Damaged[a]	During ripening: Slight	During ripening: Moderate	During ripening: Severe	During ripening: Damaged[a]
5	75	385	0	0	0	0	7.3	1.0	0.3	1.3
6	75	448	6.7	0	0	0	11.4	4.5	0.2	4.7
7	75	472	2.7	0	0	0	21.0	11.4	10.2	21.6
8	75	420	0	0	0	0	24.8	5.2	2.1	7.3
9	75	508	0	0	0	0	10.6	0.8	0	0.8
10	75	467	0	0	0	0	7.9	2.6	0.2	2.8
11	75	492	0	0	0	0	19.9	7.7	3.7	11.4
Total	525	3192								
Average			1.3	0	0	0	14.8	4.8	2.4	7.2

[a]Slight scald was not considered of commercial significance; moderate and severe scald was considered to damage the fruit and to be scorable against grade standards.

MARKET PATHOLOGY LABORATORY, BIOLOGICAL SCIENCE BRANCH, UNITED STATES DEPARTMENT OF AGRICULTURE, NEW YORK, NEW YORK

PEAR DECLINE IN THE PACIFIC NORTHWEST[1]

C. G. Woodbridge, E. C. Blodgett and T. O. Diener[2]

Abstract

In Washington and British Columbia a problem in pear culture of considerable economic importance is referred to as pear decline. Two types, designated as "quick decline" and "slow decline", are easily distinguishable. The former is characterized by a rapid wilting of the leaves followed by death of the affected tree; the latter by a non-fatal loss of vigor and reduced growth over a period of years. The cause of pear decline is not known.

Within the last few years a problem in pear culture has become serious in the Pacific Northwest. It is commonly called pear decline. Two types are easily distinguishable. A sudden dying of trees is designated "quick decline" and a gradual loss of vigor is called "slow decline". It is not known whether both conditions have the same cause. For convenience, the two declines are described together in this paper.

Both conditions are found east of the Cascade Mountains in Washington and in British Columbia, Canada. Quick decline has also been reported from the Medford area in Oregon (2). In 1949, a report (1) of work to date was presented by a commission investigating a pear "decline"problem in two provinces in Northern Italy. It was stated that the disorder, which has symptoms somewhat similar to those of slow decline, occurred as early as 1908.

Preliminary reports (2, 3, 4, 5) by workers in Washington have appeared elsewhere. One recent paper (6) details work on isolations from roots of affected and healthy pear trees. It is the purpose of this report to describe the symptoms, to indicate the importance of decline and to state briefly what investigations of this problem are underway in the State of Washington.

SYMPTOMS

Quick decline (Fig. 1) is less prevalent but is more spectacular. During the growing season a normal, vigorous tree wilts and declines rapidly. This may happen at any time before or after harvest, but this decline is more common shortly before. The leaves wilt, become dry and usually turn dark on the tree. Young wood and bark shrivel and the fruit becomes soft within a few days. The conductive tissues appear drier than normal. No other symptoms have been detected.

In several tests, branches cut from affected trees just as the wilt was starting were placed in water. These branches absorbed water and leaves again became turgid. This result suggests that the trouble might be within the root or at the stock-scion union.

In 1956 several less typical cases were noted. These were characterized by defoliation, the amount depending upon the time of the season but greater near autumn.

Vigorous trees, 2-years-old and older, have been affected with quick decline but mature trees are more commonly involved. Quick decline was observed occasionally in 1948 but has now become more prevalent. On affected trees fewer fibrous roots were found than on healthy trees. Large roots have generally appeared normal but observations in 1956 showed that some had necrotic areas. Trees showing quick decline may be scattered throughout an orchard or may occur in small groups. Sometimes three or four adjacent trees in a row are affected. The trouble has been observed on Bartlett, Anjou, Bosc, Winter Nelis, and less commonly on Flemish Beauty. Affected trees do not recover.

Slow decline (Fig. 2) develops over a period of years. In its early stages terminal growth is lessened but the tree usually sets and matures a normal crop. Moderately affected trees may bloom heavily, and have a fair set of fruit, but the tree does not have enough vigor to size the crop. In advanced stages little or no terminal growth is made; the leaves are small and

[1] Scientific Paper No. 1615, Washington Agricultural Experiment Stations, Pullman, Washington; Project No. 1337.

[2] Associate Horticulturist, State College of Washington, Pullman, Washington, Plant Pathologist, State College of Washington and Washington State Department of Agriculture, Irrigation Experiment Station, Prosser, Washington; and Associate Plant Pathologist, Irrigation Experiment Station, Prosser, Washington, respectively.

FIGURE 1. Quick decline of Bartlett: Left -- Early stage showing current season's growth on what was an apparently normal tree. Right -- A later stage. Most leaves have fallen and many unsized fruits remain on the tree. These trees did not recover.

FIGURE 2. Left -- Slow decline of Bartlett. Note the sparseness of the foliage and the lack of terminal growth. This condition develops over a number of years. Right -- A healthy Bartlett pear tree.

Table 1. The percentage of quick and slow decline in some selected pear orchards in the Yakima Valley, Washington, 1956.

Orchard	Number of trees	Percent healthy*	Percent quick decline**				Percent slow decline***			
			a	b	c	d	2	3	4	5
1	56	1	-	-	-	-	25	36	22	16
2	91	18	3	6	-	-	20	26	18	9
3	265	46	-	-	-	-	23	18	6	7
4	451	3	-	-	-	-	9	30	21	37
5	615	26	1	4	1	2	27	29	8	2
6	1,169	23	2	4	2	4	20	30	12	3

* Includes young non-bearing trees.

**a. Small fruits on tree, i. e. death approximately one month before harvest.
b. Large fruits on tree, i. e. death shortly before harvest.
c. No fruit on tree, i. e. death after harvest.
d. Doubtful. Not all typical symptoms present.

***2. Terminal growth somewhat limited.
3. Terminal growth definitely limited.
4. No growth.
5. No growth, very sparse foliage. Some trees nearly dead.

pale. The over-all appearance of the tree is one of sparseness. In advanced cases some branches may die back. Affected trees have failed to respond to good management practices. As with quick decline there are fewer fibrous roots but there does appear to be ample moisture in the conductive tissues. Affected trees are sometimes found scattered in orchards, sometimes in small groups, and occasionally several may be adjacent in rows. The trees do not die, but rather "hang on" with their appearance varying but little from year to year. Slow decline is found more commonly on Bartlett but has been observed on Anjou, Winter Nelis, Bosc, and rarely on Flemish Beauty.

In Washington, the older pear orchards were planted shortly after 1900 and are principally on imported French pear understock, Pyrus communis. Plantings of 20 to 35 years ago are mainly on Japanese understock, P. serotina. Recent plantings have used domestic Bartlett seedlings as understock. Since decline has appeared most frequently on 20-35-year-old trees, Japanese rootstock may be a factor in decline.

Table 1 summarizes the results of a survey made in some orchards in the Yakima Valley in Washington. The results show that many of the trees in the orchards examined are in a greatly reduced state of vigor. In one orchard, 12 percent of the trees were affected with quick decline. In another over 50 percent were in advanced stages (4 and 5, Table 1) of slow decline. This survey does not represent the damage to all commercial pear orchards in Washington but does indicate the importance and severity of pear decline, particularly since these disorders seem to be increasing. Other factors have curtailed production in recent years -- for example, spring frost, fire blight, winter injury, and poor pollination. Decreased production is not due entirely to decline. However, it is estimated that decline has decreased production by as much as one-third in some orchards.

Studies on both quick decline and slow decline have been underway for several years but the cause or causes remain obscure. No effective control measures are known to the authors. Studies dealing with fungi associated with pear roots (5, 6) appeared to give no correlation between the presence of these fungi and slow decline. Other studies include examination for nematodes, leaf analyses, inarching, use of various rootstocks, water management (4), tests for the presence of viruses and possible insect vectors. Stock-scion compatibility, the effect of pesticidal sprays, and winter injury are being investigated.

ACKNOWLEDGMENTS

The authors appreciate the help received from members of the Pear Decline Committee,

other agricultural research workers in Washington, and for financial assistance from the Washington State Soft Fruit Commission and from the Northwest Canners' Association.

Literature Cited

1. BALDACCI, ELIO, GIULIO CATONI, RAFFAELE CIFERRI, and LUIGI MARIMPIETRI. 1949. Relazione sui dati e le esperienze circa la "moria" del pero nella Venezia Tridentina (Trentino ed Alto Adige). Notiz. Mal. Piante No. 4, 1-51, September.
2. BLODGETT, E. C. 1956. Pear decline work in progress. Western Fruit Grower 10(12): 20-24.
3. BLODGETT, E. C., C. G. WOODBRIDGE, NELS BENSON, and W. J. O'NEILL. 1955. Report of pear decline committee. Proc. Washington State Hort. Assoc. 51st Ann. Meet. pp. 231-233.
4. DEGMAN, E. S. 1954. Is pear decline caused by over-irrigation? Proc. Washington State Hort. Assoc. 50th Ann. Meet. pp. 66-67.
5. SPRAGUE, RODERICK. 1955. Fungi isolated from the roots of pear trees at Wenatchee. Proc. Washington State Hort. Assoc. 51st Ann. Meet. p. 93.
6. SPRAGUE, RODERICK. 1957. Fungi isolated from underground parts of pear trees. Plant Dis. Reptr. 41: 74-76.

WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PULLMAN, WASHINGTON

PRODUCTION OF VIRUS-FREE FOUNDATION STRAWBERRY STOCKS IN A SCREENHOUSE

J. Bert Smith and Geo. M. Darrow[1]

For the past 5 years a screenhouse, 20 x 200 x 7 feet, has been used at Beltsville, Maryland for the propagation of foundation stock of virus-free strawberries. High yield of plants and freedom from aphids, the virus vectors, indicate that this is a very satisfactory way of growing limited quantities of foundation stocks. The soil in the screenhouse was fumigated in 1952 and 1955 with methyl bromide to destroy root nematodes, weed seeds, and soil insects, before it was planted with indexed virus-free plants. In 1955 it was planted with hot-water-treated plants that had been started in the greenhouse. Isolation has been sufficient to avoid leaf spots as a problem. No red stele appeared, even though the soil is rather heavy and the seasons ideal for its development. Mildew was more severe in the screenhouse than in the open field and reduced production of plants of the most susceptible varieties. One report on varietal resistance to mildew has been made[2]. During the first two seasons (1952 and 1953) an infestation of 2-spotted spider mites occurred in at least one variety but was eradicated by Aramite. Cyclamen mite infested one variety in 1954 and plants in the infested and adjacent

Table 1. Strawberry plants produced per block of 32 square feet (4 feet x 8 feet) by two mother plants.

Variety	Number of plants			
	1954	1955	1956	Average for 2 or 3 years
Albritton	139	32	55	75
Armore	340	75	125	180
Aroma	455	275	-	365
Bellmar	575	525	464	521
Blakemore	400	315	215	310
Catskill	255	100	321	225
Dixieland	320	110	240	223
Dunlap	695	-	-	-
Fairfax	420	-	183	301
Gem	500	-	282	391
Howard 17	345	190	272	269
Klondike	600	250	-	425
Klonmore	615	-	-	-
Marshall	210	250	-	230
Massey	375	290	-	333
Midland	344	72	200	205
Missionary	-	475+	375	-
Pocahontas	175	475	263	304
Redstar	119	-	130	125
Robinson	668	825	500	664
Sioux	400	-	-	-
Sparkle	124	200	256	193
Tennessee Beauty	345	300	450	365
Vermilion	295	200	-	247

+Not all dug.

[1] Respectively, Horticulturist, and Collaborator, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2] Darrow, Geo. M., D. H. Scott, and A. C. Goheen. 1954. Relative resistance of strawberry varieties to powdery mildew at Beltsville, Maryland, 1954. Plant Dis. Reptr. 38: 864-866.

FIGURE 1. Interior of 20 x 200-foot screenhouse, showing plant production of foundation stock of strawberries. Howard 17 (left) produced 320 plants per block (4 x 8 feet), while Armore (right) produced 315 plants.

FIGURE 2. Close view inside screenhouse showing separation of strawberry varieties by narrow alleys. Marshall produced 185 plants per block and Blakemore 400 plants.

blocks were fumigated with methyl bromide. Heat treatment for nematodes eliminated all spider and cyclamen mites. Plants in one plot had a few root-knot nematodes in 1955. The soil of that particular plot was fumigated in the spring of 1956 with methyl bromide and later replanted. At the end of 1956, 4 varieties showed a very few root-knot nematodes.

Birds became such a serious pest in 1955, by tearing holes in the screen, that the saran screen had to be replaced with fiber-glass screen in the fall of 1956. The 20 x 20 mesh screen has been sufficiently fine to keep out aphids of the virus-carrying species. Because of shading by the screen and reduced air movement, the soil was moist throughout the seasons. Only one irrigation has been given the soil in 5 years and that may not have been essential.

The ground area in the screenhouse is laid out in 5 x 10-foot blocks, with two plants set in each block. Each block of plants has been kept separate by alleys so that the actual size of the blocks has been about 4 x 8 feet, or about 32 square feet. In this screenhouse each block has been set to a separate variety or clone. The stands, including all small plants, obtained in these blocks are given in Table 1.

Because the plants were set at different times and because of other factors, records are not fully comparable for the different years. However, the records do show what may be expected in a screenhouse.

For 2 or 3 years these 24 varieties averaged 75 for Albritton to 664 per block for Robinson, or 102,075 to 906,049 plants per acre. By eliminating some of the alleys and borders, when fewer varieties and blocks are used, the plant yield could be increased. A 1/100-acre screenhouse (about 20 x 20 feet) should produce 4,000 to 8,000 plants, or enough to set about 1 to 2 acres in the open field if the plant spacing is 3 x 3 1/2 feet.

In 1957 the plants for the screenhouse were again heat-treated because of the presence of a few nematodes and will be planted later. Such heat-treated plants are potted in sterilized soil and grown for a month in the greenhouse until the screenhouse soil has been fumigated and is ready to plant.

About 25 screenhouses are now in use by strawberry plant propagators in the United States, as a safeguard for maintenance of virus-free plants. They range in size from one-half acre to a few square feet.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

COMBATING PLANT VIRUS DISEASES WITH A NEW ANTIVIRAL AGENT, CYTOVIRIN[1]

Reed A. Gray

Summary

Local lesion formation by southern bean mosaic virus and tobacco mosaic virus was completely prevented by spray levels of 0.5 to 1 ppm of crystalline Cytovirin. Remarkable protection from systemic infections caused by tomato spotted wilt virus and tobacco mosaic virus resulted when tomato and tobacco plants were sprayed twice with a solution containing 100 ppm of the crude antiviral agent.

Generally antibiotics that have been isolated because of their antibacterial or antifungal activity have not been effective against plant virus diseases as shown by Manil (4), Fulton (2), Miller and Vaughan (5), and Beale and Jones (1). Noformicin, an antibiotic that was isolated because it had antiviral activity in animals, was reported by Gray (3) to inhibit two viruses in intact plants, but its effect was not very lasting. This report is concerned with an antiviral agent that was isolated solely for its plant antiviral activity. It proved to be the most potent agent encountered as yet for combating local lesion and systemic plant virus infections.

The antiviral agent, Cytovirin, was isolated in the Merck Sharp & Dohme Research Laboratories[2] from the culture filtrates of an unidentified Streptomyces species. This paper summarizes preliminary results on its effect on local lesion production by southern bean mosaic virus and tobacco mosaic virus as well as systemic infections with these same viruses.

LOCAL LESION TESTS

The local lesion and systemic virus tests for southern bean mosaic virus and tobacco mosaic virus were carried out as described earlier by Gray (3). Using the local lesion test with southern bean mosaic virus on Pinto bean plants, it was found that the culture filtrate diluted 1:3 with water completely prevented the development of local lesions on the leaves that were sprayed. The number of lesions on the untreated primary leaves opposite the treated leaves was also reduced. Some injury to the treated leaves occurred. Further dilutions of the fermentation broth were tested. High dilutions completely prevented lesion production without injury to the treated leaves.

The fermentation broth was subjected to several chemical fractionations and crude Cytovirin of approximately 5 percent purity was prepared. Subsequently, Cytovirin was isolated in pure crystalline form. Crude and crystalline material were tested against local lesion production by southern bean mosaic virus. Pure Cytovirin was also tested against local lesion production by tobacco mosaic virus in Nicotiana rustica plants. In the latter test, all the leaves were rubbed with carborundum and tobacco mosaic virus and half of each leaf was sprayed with the antibiotic solution 1 hour later. Typical results obtained in the local lesion tests are reported in Table 1.

In local lesion tests where the leaves were injured by rubbing with carborundum, phytotoxicity was produced by low levels of the antiviral agent that did not injure unrubbed plants. Spray concentrations as low as 1 ppm of the pure antiviral agent completely prevented lesion formation by southern bean mosaic virus, and a spray level of 0.5 ppm completely prevented local lesion production by tobacco mosaic virus.

SYSTEMIC VIRUS TESTS

Systemic virus infections rather than local lesion infections cause the large losses to agricultural crops. The usefulness of crude and crystalline Cytovirin to combat systemic

---

[1] Contribution from the Merck Sharp & Dohme Research Laboratories, Division of Merck & Co., Inc. Rahway, New Jersey.

[2] The author is indebted to Mr. R. A. Long and Dr. H. B. Woodruff for the microbiological production and to Mr. F. R. Koniuszy, Dr. E. Kaczka and Dr. Karl Folkers for the chemical isolation.

Table 1. The effect of Cytovirin on local lesion formation by two viruses.

Cytovirin Spray Treatment	Lesion Ratio Treated : Untreated Southern Bean Mosaic Virus[a]	% Reduction In Lesions Caused by Cytovirin	Phyto-toxicity
100 ppm. crude	0:157	100	slight
20 ppm. crude	22:239	93	slight
4 ppm. crude	53:246	78	none
1 ppm. pure	0:188	100	slight
0.5 ppm. pure	21:202	90	none
	Tobacco Mosaic Virus[b]		
0.5 ppm. pure	1:263	100	slight
0.25 ppm. pure	19:444	96	none
0.12 ppm. pure	12:263	95	none

a. Ratio of the number of virus lesions on 4 treated bean leaves to number on opposite four untreated primary leaves for each treatment.

b. Ratio of number of virus lesions on 6 treated N. rustica half-leaves to number on 6 opposite half-leaves for each treatment.

Table 2. Effect of Cytovirin against two systemic plant virus diseases.

Cytovirin Spray Treatment	Ratio of No. of Plants Showing Virus Symptoms to No. of Plants in Treatment Weeks After Inoculation						Avg. Ht. of Plants 4 Weeks
	2	3	4	5	6	7	mm.
	Tobacco mosaic in tobacco:						
0 untreated controls	7:7	7:7	7:7	7:7	7:7	7:7	414
100 ppm. crude, 2 hrs. after inoc. and again 12 days later.	2:7	2:7	2:7	3:7	3:7	3:7	613
	Spotted wilt in tomato:						
0 untreated controls	8:8	8:8	8:8	8:8	8:8	8:8	213
100 ppm. crude 2 hrs. after inoc. and again 12 days later	2:8	2:8	2:8	2:8	2:8	2:8	506

virus infections in plants grown in the greenhouse was investigated. The systemic virus infections examined were tobacco mosaic virus in White Burley tobacco plants and spotted wilt virus in Bonny Best tomato plants.

The third leaf from the top of each tomato and tobacco plant was sprinkled with carborundum and rubbed with the appropriate virus suspension when the plants were 8 to 10 inches tall. In the case of spotted wilt virus, the undiluted juice from infected tomato leaves was used for the inoculum[3]. The inoculum of tobacco mosaic virus used was capable of producing 200 lesions per leaf on N. rustica plants. Half of the tomato and tobacco plants were sprayed on the upper and lower leaf surfaces with an aqueous solution containing 100 ppm of crude Cytovirin 2 hours after inoculation and again 12 days later. The other half of the inoculated plants remained untreated as controls. The plants were observed carefully for virus symptoms at regular intervals, and the results are summarized in Table 2.

The results reported in Table 2 show that spraying crude Cytovirin on the plants 2 hours after inoculation and again 12 days later prevented the development of tobacco mosaic virus and spotted wilt virus in most of the treated tobacco and tomato plants for as long as 7 weeks. In each case the two treated plants that became diseased in 2 weeks showed virus symptoms when the second spray was applied 12 days after inoculation. It appeared that, once the virus became thoroughly established throughout the plant, spraying with Cytovirin would not cure the disease. The effect of the antiviral agent in preventing stunting by the two viruses was remarkable.

Other experiments are in progress which show that Cytovirin is active against other plant viruses. Experiments to be reported later also show that it can be applied as long as 1 day before inoculation or as long as 1 day after inoculation and still exert pronounced protective action against systemic plant virus diseases.

Literature Cited

1. BEALE, HELEN PURDY, and C. R. JONES. 1951. Virus disease of tobacco mosaic and potato yellow dwarf not controlled by certain purified antibiotics. Boyce Thompson Inst. Contr. 16: 395-407.
2. FULTON, R. H. 1954. Studies on inactivation of the strawberry type 2 virus in vivo. (Abstr.) Phytopathology 44: 489.
3. GRAY, R. A. 1955. Activity of an antiviral agent from Nocardia on two viruses in intact plants. Phytopathology 45: 281-285.
4. MANIL, P. 1947. Action negative, sur le virus de la mosaique du tabac, de la penicillini et du prontosil. Ann. Inst. Pasteur. 73: 294.
5. MILLER, P. W., and E. K. VAUGHAN. 1957. Yellows virus complex in Marshall strawberry plants not inactivated by growth regulating, antibiotic, and certain other chemicals. Plant Dis. Reptr. 41: 17-18.

MERCK SHARP & DOHME RESEARCH LABORATORIES, MERCK & CO., INC., RAHWAY, NEW JERSEY

[3] The initial inoculum for spotted wilt virus was kindly supplied by Dr. B. H. Davis of Rutgers University.

A BIBLIOGRAPHY OF VIRUSES INFECTING THE SOYBEAN (GLYCINE MAX (L.) MERR.)

K. W. Kreitlow[1], Helen C. Boyd[2], D. W. Chamberlain[1], and John M. Dunleavy[3]

Virus diseases of soybeans have become increasingly prevalent and damaging during the past several years. The majority of references pertaining to virus diseases of this host prior to 1950 were listed in Ling's Bibliography of Soybean Diseases published in 1951. Since then additional research has been conducted on viruses that affect legumes, many of which are also capable of infecting soybeans. It was felt that a compilation of such references to date might be of assistance not only to plant pathologists investigating virus diseases of soybeans but to pathologists investigating other virus diseases as well. Although considerable effort was made to locate all references dealing with viruses infectious to soybeans, it is possible that some applicable references were inadvertently overlooked. This is particularly true for papers published in journals that are not readily available.

Some of the references listed concern viruses that have not yet been found infectious to soybeans in nature but that can be transmitted experimentally to this host. Although such viruses may seldom attack soybeans in nature they should be considered potentially infectious and should not be ignored.

Most of the references listed were checked in the original publication or were obtained as citations from at least two sources. Those marked with an asterisk were obtained from a single source and could not be verified further.

AFANASIEV, M. M., and H. E. MORRIS. 1952. Bean virus 2 (yellow) on Great Northern Bean in Montana. Phytopath. 42: 101-104.

AITKEN, Y., and B. J. GRIEVE. 1943. A mosaic virus of subterranean clover. Australian Inst. Agr. Sci. J. 9: 81-82.

ALBRECHT, H. R. 1939. Soybean virus disease studies. Alabama Agr. Expt. Sta. 50th. Ann. Rpt. p. 19.

ALLINGTON, W. B. 1944. Soybean disease investigations at the U. S. Regional Soybean Laboratory. Soybean Digest 4 (11): 60, 65.

ALLINGTON, W. B. 1946. Soybean diseases in the Cornbelt Region in 1945. Soybean Digest 6 (11): 48.

ALLINGTON, W. B. 1946. Bud blight of soybean caused by the tobacco ringspot virus. Phytopath. 36: 319-322.

ANNUAL REPORT CANADIAN PLANT DISEASE SURVEY. Vols. 13-34 (1933-1954). Science Service, Botany and Plant Pathology Division, Canada Department of Agriculture.

ANONYMOUS. 1932. Virus diseases of tobacco in Nyasaland. Nyasaland Dept. Agr. Bul. 2. 15p.

ANONYMOUS. 1933. Progress made in study of bean diseases. Wis. Agr. Expt. Sta. Bul. 425: 101-102.

ANONYMOUS. 1943. Reports on diseases of soybeans. Plant Dis. Rptr. 27: 299-301.

ANONYMOUS. 1943. Reports on diseases of soybeans. Plant Dis. Rptr. 27: 506-514.

Respectively, [1]Plant Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture; [2]Librarian, Beltsville Branch, United States Department of Agriculture Library; [3]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Iowa Agricultural Experiment Station cooperating.

ANONYMOUS. 1943. Reports on diseases of soybeans. Plant Dis. Rptr. 27: 603-606.

ANONYMOUS. 1944. Reports on soybean diseases. Plant Dis. Rptr. 28: 752-753.

ANONYMOUS. 1944. Other reports on soybean diseases. Plant Dis. Rptr. 28: 959-960.

ANONYMOUS. 1948. Plant diseases. Notes contributed by the Biological Branch. Agr. Gaz. New South Wales 59: 527-536.

ANONYMOUS. 1952. Diseases in the Minnesota-Iowa-Missouri area. Soybean Digest 12 (9): 15.

ANONYMOUS. 1953. Distribution, symptoms and control of some of the more important plant diseases. Plant Dis. Rptr. Suppl. 221: 107-181.

ATANASOFF, D. 1934. Virus diseases of plants: A bibliography. 219p.

ATANASOFF, D. 1937. Virus diseases of plants: A bibliography. Suppl. I. Phytopathologische Zeitschrift 10: 339-463.

ATANASOFF, D. 1940. Virus diseases of plants: A bibliography. Suppl. II. Phytopathologische Zeitschrift 12: 511-584.

ATHOW, K. L, and A. H. PROBST. 1952. Indiana soybean disease survey 1952. Purdue Agr. Expt. Sta. Mimeo. BP-52. (7 p.)

ATKINSON, R. E. 1944. Soybean diseases in Virginia and West Virginia. Plant Dis. Rptr. 28: 1008.

BAIN, D. 1944. Diseases on soybean in Louisiana. Plant Dis. Rptr. 28: 540-541.

BAIN, DOUGLAS C. 1945. Soybean mosaic in Louisiana. In Diseases reported on leguminous forage and cover crops. Plant Dis. Rptr. 29: 495.

BALDACCI, E. 1949. Soybean mosaic in Italy. (In Italian.) Olearia 3: 111-112.

BAUDYS, E. 1931. (Phytopathological Notes VII.) Ochrana Rostlin 11: 178-197. (In Czechoslovakian.)

BAWDEN, F. C. 1956. Plant viruses and virus diseases. 3d ed. 335p. Waltham, Mass., Chronica Botanica.

BAWDEN, F. C., R. P. CHAUDHURI, and B. KASSANIS. 1951. Some properties of broad bean mottle virus. Ann. Appl. Biol. 38: 774-784.

BERKELEY, G. H. 1947. A strain of the alfalfa mosaic virus on pepper in Ontario. Phytopath. 37: 781-789.

BOYD, O. C. 1943. Diseases in Massachusetts in 1942. Plant Dis. Rptr. 27: 96-99.

BRETZ, T. W. 1943. Surveys in Iowa and Missouri. Plant Dis. Rptr. 27: 377-380.

BRETZ, T. W. 1944. Summary of plant diseases observed in Iowa during 1943. Plant Dis. Rptr. Suppl. 147: 223-224.

BRETZ, T. W. 1944. Summary of plant diseases observed in Missouri during 1943. Plant Dis. Rptr. Suppl. 148: 294-302.

BRETZ, T. W. 1944. Diseases observed on soybeans in Missouri. Plant Dis. Rptr. 28: 832-834.

BRETZ, T. W. 1944. Diseases in Missouri. Plant Dis. Rptr. 28: 891.

BRIDGMON, G. H., and J. C. WALKER. 1952. Gladiolus as a virus reservoir. Phytopath. 42: 65-70.

BROWN, W., F. T. BROOKS, and F. C. BAWDEN. 1948. A discussion on the physiology of resistance to disease in plants. Roy. Soc., London. Proc., Ser. b., Biol. Sci. 135: 171-195.

CALVINO, E. M. 1942. Virosi bellosa e mosaico internervale in diverse varieta di Soja. Costa Azzura Agric. Flor., Sanremo. 22: 85-87.

CASTELLANI, E. 1948. Le virosi della soia. Olearia 2: 838-844.

CASTELLANI, E. 1950. Su alcune virosi delle leguminose foraggere osservate in provincia di Firenze. (On some virus diseases of forage legumes observed in the province of Florence.) Notiz. Malatt. Piante 12: 1-8.

CHAMBERLAIN, D. W. 1948. Soybean disease investigations in 1947. Soybean Digest 8(10): 18.

CHAMBERLAIN, D. W., and B. KOEHLER. 1951. Soybean diseases in Illinois. Ill. Agr. Expt. Sta. Cir. 676. 31p.

CHAMBERLAIN, E. E. 1939. Pea-streak (Pisum virus 3). New Zealand J. Sci. Tech. 20A: 365-381.

CHESTER, K. S., and W. E. COOPER. 1944. A lethal virus of guar (Cyamopsis psoraloides DC.). Phytopath. 34: 998.

*CHU, C. R. 1941. (Observations on plant diseases in Sikiang.) (In Chinese.) In the Report on the Scientific Expedition to the Province of Sikiang, by the Chinese Natural Sciences Society. p. 93-115.

CLINTON, G. P. 1916. Notes on plant diseases of Connecticut. In Conn. Agr. Expt. Sta. Ann. Rpt. 1915: 446-447.

CONOVER, R. A. 1948. Studies of two viruses causing mosaic diseases in soybean. Phytopath. 38: 724-735.

COOK, M. T. 1935. Host index of virus diseases of plants. Puerto Rico Univ. J. Agr. 20: 315-406.

COOPER, W. E. 1949. Top necrosis, a virus disease of guar. Phytopath. 39: 347-358.

CRALL, J. M. 1950. Soybean diseases in Iowa in 1949. Plant Dis. Rptr. 34: 96-97.

CRALL, J. M. 1951. Soybean diseases in Iowa in 1950. Plant Dis. Rptr. 35: 320-321.

CRALL, J. M. 1952. Soybean diseases in Iowa in 1951. Plant Dis. Rptr. 36: 302.

DALE, W. T. 1943. Preliminary studies of the plant viruses of Trinidad. Trop. Agr. (Trinidad) 20: 228-235.

DANA, B. F. 1939. Occurrence of curly top in the Pacific Northwest in 1938. Plant Dis. Rptr. 23: 107-113.

DANA, B. F. 1940. Occurrence of big bud of tomato in the Pacific Northwest. Phytopath. 30: 866-869.

DANA, B. F. 1941. Morphological and anatomical features of phyllody in varieties of tomatoes and beans. Phytopath. 31: 168-175.

DANA, B. F. 1947. Phyllody of common beans, a graft-transmissible disease. Phytopath. 37: 360-361.

DESJARDINS, P. R., R. L. LATTERELL, and J. E. MITCHELL. 1954. Seed transmission of tobacco-ringspot virus in Lincoln variety of soybean. Phytopath. 44: 86.

DICKSON, B. T. 1924. Mosaic studies IV. Phytopath. 14: 346.

DOUNINE, M. S., ed. 1938. (Virus diseases of plants.) II. Virusnye-boleznirastenii. 240p. Moskva: Vsesoyuzu. Akad. Selskhoz. Nauk Lenina, Vol. 2. Virus diseases of the soybean, by E. D. Yakimovich, p. 226, 227.

DUNLEAVY, J. M. 1954. Soybean diseases in Iowa in 1953. Plant Dis. Rptr. 38: 89-90.

DUNLEAVY, J. M. 1955. Soybean diseases in Iowa in 1954. Plant Dis. Rptr. 39: 169-170.

FENNE, S. B. 1942. More about soybean diseases from Virginia. Plant Dis. Rptr. 26: 382.

FRANDSEN, N. O. 1952. Untersuchungen zur Virusresistenzzuchtung bei Phaseolus vulgaris L. I. Phytopathologische Untersuchungen. (Studies on breeding for virus resistance in Phaseolus vulgaris L. I. Phyto-pathological studies.) Ztschr. f. Pflanzenzucht. 31: 381-426.

FROMME, F. D. 1921. Diseases of cereal and forage crops in the United States in 1920. Plant Dis. Rptr. Suppl. 15: 173.

FULTON, R. W. 1948. Hosts of the tobacco streak virus. Phytopath. 38: 421-428.

GARDNER, M. W. and J. B. KENDRICK. 1921. Soybean mosaic. J. Agr. Res. 22: 111-114.

GRANCINI, P. 1941. Il "mosaico" della soja. Avvenire Agr., Milano, 49: 231-235.

HAENSELER, C. M. 1946. Soybean diseases in New Jersey. N. J. Agr. Expt. Sta. Plant Disease Notes. 23: 17-20.

HAENSELER, C. M. 1947. Pathologist describes four principal diseases of field soybeans in Jersey. New Jersey Agr. 29(3): 4.

HAGEDORN, D. J. 1950. A cucumber virus strain with a wide leguminous host range. Phytopath. 40: 11.

HAGEDORN, D. J., and J. C. WALKER. 1949. Wisconsin pea streak. Phytopath. 39: 837-847.

HAGEDORN, D. J., and J. C. WALKER. 1950. The relation of bean virus 2 to pea mosaic in Wisconsin. Phytopath. 40: 684-698.

HANSFORD, C. G. 1934. Annual report of mycologist, 1933. Uganda. Dept. Agr. Annual Rpt. 1933, pt. 2, p. 48-51.

HARA, K. 1930. Pathologia agriculturalis plantarum. (In Japanese.) 950p. Tokyo.

HEINZE, K. 1941. Feldinfektionsversuch mit dem Sojabohnenvirus. Berlin. Biol. Reichsanst. f. Land-u. Forstw. 65: 23.

HEINZE, K. 1942. Feldbereinigungen bei Sojakulturen als Schutzmassnahme gegen die Ausbreitung des Virosen Sojamosaiks. Züchter 14: 254-258.

HEINZE, K., and E. KÖHLER. 1940. Die Mosaikkrankheit der Sojabohne und ihre Übertragung durch Insekten. Phytopath. Ztschr. 13: 207-242.

HILDEBRAND, A. A. 1944. A symposium. (On seed-borne diseases.) Canad. Phytopath. Soc. Proc. 12: 18-21.

HILDEBRAND, A. A. 1948. Keeping abreast with soybean diseases in Ontario. Soybean Digest 8(10): 16-17.

HILDEBRAND, A. A., and L. W. KOCH. 1947. Observations on bud blight of soybeans in Ontario. Sci. Agr. 27: 314-321.

HOLMES, F. O. 1939. Handbook of phytopathogenic viruses. 221p. Minneapolis, Burgess.

HOLMES, F. O. 1948. The filterable viruses. Suppl. 2 in Bergey's Manual of Determinative Bacteriology, VI ed. p. 1127-1286. Baltimore, Md. Williams and Wilkins.

HONEY, E. E. 1944. Soybean diseases in Wisconsin. Plant Dis. Rptr. 28: 871-872.

HONEY, E. E., J. G. DICKSON, and F. R. JONES. 1944. Diseases of soybeans in Wisconsin. Plant Dis. Rptr. 28: 988-990.

HOWARD, F. L. 1942. An undetermined, apparently virus, disease of edible soybeans in Rhode Island. Plant Dis. Rptr. 26: 381.

*IWADARE, S., M. SASAKI, and N. NAITO. 1943. Catalogue of diseases of farm crops in "Manchukuo". (In Japanese.) "Manchukuo" Nat. Agr. Expt. Sta. Rpt. No. 45. 233p.

JOHNSON, F. 1943. Soybean streak in Ohio. Plant Dis. Rptr. 27: 86-87.

JOHNSON, H. W., and D. W. CHAMBERLAIN. 1953. Bacteria, fungi, and viruses on soybeans. USDA Yearbook 238-247.

JOHNSON, H. W., D. W. CHAMBERLAIN, and S. G. LEHMAN. 1954. Diseases of soybeans and methods of control. USDA Cir. No. 931. 40p.

JOHNSON, H. W., D. W. CHAMBERLAIN, and S. G. LEHMAN. 1955. Soybean diseases. USDA Farmers' Bul. No. 2077. 16p.

JOHNSON, H. W., and R. A. KILPATRICK. 1953. Soybean diseases in Mississippi in 1951-1952. Plant Dis. Rptr. 37: 154-155.

JOHNSON, H. W., and B. KOEHLER. 1943. Soybean diseases and their control. USDA Farmers' Bul. No. 1937.

JOHNSON, J. 1922. Relation of air temperature to the mosaic disease of potatoes and other plants. Phytopath. 12: 438-440.

JOHNSON, J. 1942. Studies on the viroplasm hypothesis. J. Agr. Res. 64: 443-454.

KAHN, R. P. 1956. Seed transmission of tomato ringspot virus in the Lincoln variety of soybean. Phytopath. 46: 295.

KAHN, R. P., and F. M. LATTERELL. 1955. Symptoms of bud blight of soybeans caused by the tobacco- and tomato-ringspot virus. Phytopath. 45: 500-502.

*KATEVA, O. 1932. Diseases of soy. In Summary account of Mountain Zonal Maize-Soy-Potato Experiment Station for 1931. Part III. Dept. of Phytopathology. Diseases of maize and soy under conditions of North Caucasus. Nauchn. Trudy Gorskaia Zonal. Kukur-Soevo-Kartof. Opytn. Stan. Ser. 1. 4: 79-101. (In Russian)

KENDRICK, J. B., and M. W. GARDNER. 1924. Soybean mosaic: seed transmission and effect on yield. J. Agr. Res. 27: 91-98.

KENT, G. C. 1942. Soybean diseases in Iowa. Plant Dis. Rptr. 26: 359.

KILPATRICK, R. A. 1955. Soybean diseases in the Delta area of Mississippi in 1954. Plant Dis. Rptr. 39: 578-579.

KOEHLER, B. 1930. Diseases of soybeans in Illinois. Amer. Soybean Assoc. Proc. 60-64.

KOEHLER, B. 1944. New developments in soybean disease studies. Soybean Digest 4(8): 6-7.

*KOEHLER, E., and K. HEINZE. 1941. Übertragungsversuche mit dem Mosaikvirus der Sojabohne. Landw. Jahrb. 90: 233.

KREITLOW, K. W., and W. C. PRICE. 1949. A new virus disease of ladino clover. Phytopath. 39: 517-528.

LARSH, H. W. 1944. Soybean diseases in Arkansas. Plant Dis. Rptr. 28: 870-871.

LARSH, H. W. 1944. Diseases observed in soybeans in Arkansas. Plant Dis. Rptr. 28: 956-957.

LARSH, H. W. 1944. Diseases reported on soybean in Oklahoma. Plant Dis. Rptr. 28: 1010.

LARSH, H. W. 1944. Diseases observed on soybeans in Arkansas. Plant Dis. Rptr. 28: 1125-1126.

LAVROVF, N. N. 1932. Key for the identification of vegetable parasites of cultivated and wild useful plants of Siberia. Part 1. (In Russian.) Tomsk, Izdatel'stvo "Kusuch", 148 p.

LeBEAU, F. J. 1947. A virus-induced top necrosis in beans. Phytopath. 37: 434.

LIHNELL, D. 1939. Några iakttagelser rörande sjukdomar på soja i vårt land. Statens Växtkyddsanstalt Växtskyddsnotiser 3: 69-73.

LING, LEE. 1951. Bibliography of soybean diseases. Plant Dis. Rptr. Suppl. 204: 110-173.

MANNS, T. F., and J. F. ADAMS. 1924. Dept. of Plant Path. and Soil Bacteriology, Del. Agr. Expt. Sta. Ann. Rpt. 1923: 25-48.

MARKLEY, K. S. 1951. Soybeans and soybean products. 2 vols. N.Y., Interscience. Diseases, p. 50-54.

MELHUS, I. E. 1942. Soybean diseases in Iowa in 1942. Plant Dis. Rptr. 26: 431-432.

MILLER, P. R. (compiler). 1953. Plant disease situation in the United States. F.A.O. Plant Protect. Bul. 2: 24-27.

MORSE, W. J., J. L. CARTTER, and L. F. WILLIAMS. 1949. Soybeans: culture and varieties. USDA Farmers' Bul. No. 1520. (Rev.) 38p.

MURATA, T. 1925. Soybean mosaic. (In Japanese.) J. Plant Protect. (Tokyo) 12: 451-452.

*NAKATA, K. 1940. Report of diseases of agricultural and horticultural crops in North China and Mongolia. (In Japanese.) Agr. Expt. Sta. North China Survey Rpt. 1: 1-92.

*NAKATA, K. 1941. A report on a disease survey of farm and horticultural crops in North China. (In Japanese.) North China Agr. Expt. Sta. Bul. No. 1, 72p.

*NAKATA, K., and ASUYAMA, H. (?date) Report on diseases of main agricultural crops in Manchuria. (In Japanese.) Manchuria Bur. Indus. Rpt. 32: 1-166.

PADY, S. M. 1943. Plant diseases in Kansas and Nebraska. Plant Dis. Rptr. 27: 375-377.

PADY, S. M. 1944. Notes on diseases observed in the Nebraska plant disease survey, August to November, 1943. Plant Dis. Rptr. Suppl. 149: 311-313.

PADY, S. M. 1944. Notes on the plant disease survey in Kansas, August to November, 1943. Plant Dis. Rptr. Suppl. 149: 313-316.

PERSON, L. H. 1944. List of plant diseases observed during surveys in Mississippi and Louisiana, August to November, 1943. Plant Dis. Rptr. Suppl. 148: 280-283.

PETTY, M. A. 1943. Soybean disease incidence in Maryland in 1942 and 1943. Plant Dis. Rptr. 27: 347-349.

PETTY, M. A. 1944. Soybean diseases on the eastern shore of Maryland. Del. State Board Agr. Bul. 33(5): 58-62.

PIERCE, W. H. 1934. Viroses of the bean. Phytopath. 24: 87-115.

PIERCE, W. H. 1935. The identification of certain viruses affecting leguminous plants. J. Agr. Res. 51: 1017-1039.

PINCKARD, J. A. 1942. Diseases of soybeans and peanuts in Mississippi. Plant Dis. Rptr. 26: 473.

PORTER, R. H. 1926. A preliminary report of surveys for plant diseases in East China. Plant Dis. Rptr. Suppl. 46: 153-166.

PRINCE, A. E. 1944. Soybean diseases in South Carolina. Plant Dis. Rptr. 28: 1125.

*QUANJER, H. M. 1938. Iets over de virusziekten van Tropische Cultuurgewassen. (An account of some virus diseases of tropical plants, tobacco, cassave, soja, arachnis, musa, sugarcane and rice). Landbouwk. Tijdschr., Wageningen, 50: 324-338.

QUANTZ, L. 1951. Untersuchungen über viruskrankheiten an Buschbohnen. Berlin. Biol. Zent. f. Land-u. forstwirt. Mitt. 70: 74-79.

QUANTZ, L. 1952. Die wichtigsten viruskrankheiten der heimischen Leguminosen. (The most important virus diseases of indigenous Leguminosae.) Saatgut-Wirtsch. 4(2): 34-37.

SAMSON, R. W. 1942. Tobacco ring-spot on edible soybeans in Indiana in 1941. Plant Dis. Rptr. 26: 382.

SĂVULESCU, T., C. SANDU-VILLE, T. RAYSS, and A. V. ALEXANDRI. 1935. Phyto-sanitary conditions in Rumania during the year 1933-34. Inst. Cerc. Agron. Romaniei Publ. 24: 1-59.

SĂVULESCU, T., C. SANDU-VILLE, A. ARONESCU, and A. V. ALEXANDRI. 1936. Phytosanitary conditions in Rumania during the year 1934-1935. Inst. Cerc. Agron. Romaniei Publ. 25: 1-97.

SĂVULESCU, T., A. ARONESCU, C. SANDU-VILLE, and A. V. ALEXANDRI. 1937. Phytosanitary conditions in Rumania during the Year 1935-1936. Inst. Cerc. Agron. Romaniei Publ. 38: 1-70.

SILBERSCHMIDT, K., and N. R. NOBREGA. 1942. Notas sobre uma doença de virus em feijao de porco (Canavalia ensiformis, D.C.) e outra em feijao comum (Phaseolus vulgaris L.). Biologico 8: 129-133.

SLAGG, C. M. 1944. Soybean diseases in Nebraska. Plant Dis. Rptr. 28: 1009.

SLAGG, C. M. 1944. Soybean diseases in Kansas. Plant Dis. Rptr. 28: 1126.

SMITH, K. M. 1937. A textbook of plant virus diseases. 615p. London, Churchill.

STONE, G. M., and J. L. SEAL. 1944. Plant diseases observed in Alabama in 1943. Plant Dis. Rptr. Suppl. 148: 276-280.

STUBBS, M. W. 1937. Certain viroses of the garden pea, Pisum sativum. Phytopath. 27: 242-266.

TERVET, I. W. 1943. Soybean diseases in Minnesota. Plant Dis. Rptr. 27: 135-138.

TERVET, I. W. 1943. Plant disease surveys in Minnesota and the Dakotas. Plant Dis. Rptr. 27: 373-375.

TERVET, I. W. 1944. Diseases of soybeans in Minnesota. Plant Dis. Rptr. 28: 687.

TERVET, I. W. 1944. Soybean diseases in Minnesota. Plant Dis. Rptr. 28: 835.

THOMAS, H. R. 1951. Yellow dot, a virus disease of bean. Phytopath. 41: 967-974.

THOMAS, H. R., and W. J. ZAUMEYER. 1950. Red node, a virus disease of beans. Phytopath. 40: 832-846.

THOMAS, H. R., and W. J. ZAUMEYER. 1953. A strain of yellow bean mosaic virus producing local lesions on tobacco. Phytopath. 43: 11-15.

TIDD, J. S. 1944. Bud blight, wildfire, and other diseases in Illinois. Plant Dis. Rptr. 28: 890.

TIDD, J. S. 1944. Bud blight in Indiana. Plant Dis. Rptr. 28: 891.

TIDD, J. S. 1944. Soybean diseases in Indiana and Illinois. Plant Dis. Rptr. 28: 957-958.

*WANG, C. M. 1942. Manual of the plant diseases of Honan province. (In Chinese.) 60p. Honan, China, Honan University.

WEIMER, J. L. 1947. Disease survey of soybean nurseries in the South. Plant Dis. Rptr. Suppl. 168: 27-53.

WEISS, F. 1945. Viruses described primarily on leguminous vegetable and forage crops. Plant Dis. Rptr. Suppl. 154: 32-80.

WEISS, M. G. 1949. Soybeans. Advances in Agronomy 1: 77-157.

WELCH, A. W. 1947. A study of soybean diseases and their control. Iowa Agr. Expt. Sta. Report on Agricultural research for the year ending June 30, 1947. Part 1, p. 170-171.

*YOSHI, H. 1927. Crop diseases in 1926. (In Japanese) Chosen Agr. Expt. Sta. Ann. 7: 21-34.

YU, T. F. 1939. A list of plant viroses observed in China. Phytopath. 29: 459-461.

YU, T. F. 1940. A list of important crop diseases occurring in Kiangsu province (1934-1937). Lingnan Sci. Jour. 19: 67-78.

ZAUMEYER, W. J. 1938. A streak disease of peas and its relation to several strains of alfalfa mosaic virus. J. Agr. Res. 56: 747-772.

ZAUMEYER, W. J., and L. L. HARTER. 1943. Two new virus diseases of beans. J. Agr. Res. 67: 305-328.

ZAUMEYER, W. J., and H. R. THOMAS. 1950. Yellow stipple, a virus disease of bean. Phytopath. 40: 847-859.

ZAUMEYER, W. J., and H. REX THOMAS. 1957. A monographic study of bean diseases and methods for their control. U.S.D.A. Tech. Bul. 868. 255p.

Sources Consulted:

Agricultural Index, 1928-Oct. 1956.
Bibliography of Agriculture, 1942-Oct. 1956.
Biological Abstracts, 1951-1955.
Experiment Station Record, 1930-1948.
Plant Disease Reporter, 1929-Oct. 1956.
Plant Disease Reporter Supplement 1949-July 1956.
Review of Applied Mycology, 1930-Sept. 1956.

UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND AND URBANA, ILLINOIS; UNITED STATES DEPARTMENT OF AGRICULTURE AND IOWA AGRICULTURAL EXPERIMENT STATION, AMES, IOWA.

WHEAT STREAK MOSAIC, A DISEASE OF SWEET CORN IN IDAHO[1]

A. M. Finley[2]

Summary

Wheat streak mosaic is reported to have occurred in epiphytotic proportions in sweet corn in southwestern Idaho. Tests of selected strains of the F_1 hybrid Golden Cross Bantam and related inbreds revealed a wide variation in susceptibility. It seems probable, therefore, that resistant strains can be developed.

INTRODUCTION

A report of the occurrence of virus-like symptoms on sweet corn growing near Melba, Idaho, was received at the Plant Pathology Laboratory of the Parma Branch of the Idaho Agricultural Experiment Station in July of 1951. An inspection revealed this to be an increase field for the production of the Golden Cross Bantam seed. The field comprized about 4 acres and it was estimated that approximately 75 to 80 percent of the ear parent plants were affected. A few of the pollen parent plants were affected but the incidence was insignificant. The difference in the reaction of the two inbreds was very striking and the distribution of infection among the female plants was also very striking. All of the female parents on the northern and western sides of the field exhibited symptoms but there was a progressive decrease in the number of infected plants toward the southeast corner. Other fields in the vicinity which were planted to these particular inbreds were examined and all were found to contain infected plants but the incidence of the disease was low. Infected plants could not be found in fields of other varieties.

The disease symptoms as they appeared in these fields were similar to those of corn mosaic as described by Brandes[3], but since the variety Golden Bantam is not a suscept for corn mosaic a positive diagnosis was not attempted.

SYMPTOMATOLOGY

This disease, like other mosaic viruses on corn, can be detected from relatively long distances by the pale yellow coloring of the affected plants. When the host plant is the inbred P-39, this yellowing is usually accompanied by dwarfing, but in the F_1 hybrid, Golden Cross Bantam, dwarfing is not noticeable. The earliest symptom is the appearance of numerous small chlorotic spots or broken streaks on the tips of the apical leaves (Fig. 1). As the disease progresses the streaks elongate parallel to and are bounded by the veins of the leaves. Older leaves frequently become completely chlorotic near the tips except for epidermal cells bordering the larger veins, but they do not exhibit necrosis nor do they show any tendency to split or shred. In this respect this disease differs from most of the other virus diseases reported on corn. Ear structures on infected plants are poorly developed and kernels are widely spaced. The kernels which develop appear to be normal and germinate readily.

FIELD SURVEYS

Field surveys were conducted in 1951, 1952, 1953, 1954 and 1955 to determine the prevalence, distribution and pattern of spread of the disease in seed and canning corn production fields. In 1951 the disease was found only in seed production fields in the Melba district. In 1952 the first report of its occurrence came from a field of market corn of Golden Cross Bantam growing in the vicinity of New Plymouth. Later surveys revealed the disease to be present in scattered fields throughout the Payette River Valley and in the Melba seed producing area. In 1953 and 1954 outbreaks were confined principally to the Payette River Valley, though reports of infected plants were received from such widely scattered areas as Lewiston and Salmon,

---

[1] Published with the Approval of the Director of the Idaho Agricultural Experiment Station as Research Paper Number 431.

[2] Plant Pathologist, Idaho Agricultural Experiment Station.

[3] Brandes, E. W. 1920. Mosaic disease of corn. Jour. Agr. Res. 19: 517-521.

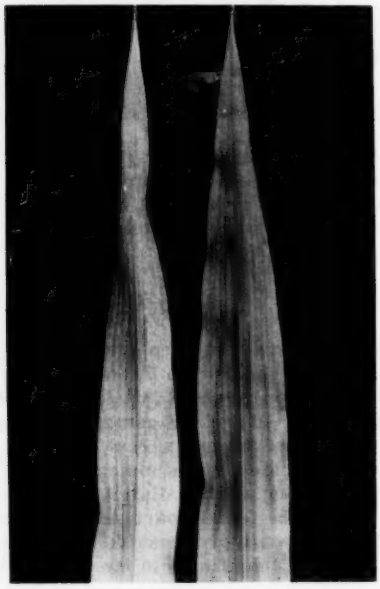


FIGURE 1. Symptoms of wheat streak mosaic on Golden Cross Bantam sweet corn. Infected leaf on left, healthy on right.

Idaho.

Infected plants were found in nearly all fields of Golden Cross Bantam but the incidence was high only in fields which were bordered by cereal, hay or pasture crops. When these crops were growing on the windward sides of the corn fields the infestation was usually severe and was always most prevalent in border rows immediately adjacent to the cereal, hay or pasture plantings. This distribution of the disease was very striking and indicated the presence of a wind-borne vector.

In 1955 the disease was found on only a few plants in the vicinity of Emmett, Idaho. This sudden decrease in prevalence might have been due to the more severe weather conditions which existed in the corn-growing areas during the winter of 1954-55. It might also have been due, in part, to the reduced acreage planted to Golden Cross Bantam.

TRANSMISSION STUDIES AND HOST RANGE

The observation of the distribution of infected plants and the association of cereal and forage crops led to the search for an insect vector and for host plants among these crops. Numerous insects were collected from infected corn plants and from bordering fields and were taken to the greenhouse at the Parma Branch Station. Transmission studies were carried out under controlled conditions but none of the insects proved capable of transmitting the disease. It must be pointed out, however, that Aceria tulipae, the known vector of wheat streak mosaic, was neither found in the field nor tested in greenhouse studies.

Surveys of wild and cultivated grass plants present in and adjacent to diseased corn fields were made. Of the plants studied, only one, Setaria viridis, exhibited disease symptoms similar to those which occurred on corn. Subsequent greenhouse tests under controlled conditions using mechanical methods of inoculation to determine host range of this virus yielded the results shown in Table 1.

The results of these tests confirmed the identity of the virus in question as that of wheat streak mosaic.

Since this disease was present in some degree in many of the seed production fields it was of vital importance to determine whether it could be transmitted through the seed and thus be perpetuated. Matured ears of grain were harvested from infected plants in 1951, 1952 and 1953. In each of the succeeding seasons approximately 1500 plants were grown from this seed in field plots at the Parma Branch Station. All of these plants appeared normal and showed no

Table 1. Host range of wheat streak mosaic virus isolated from Golden Cross Bantam sweet corn.

Species	Variety tested	Susceptible
Corn	Inbred P-39	Yes
	Inbred P-51	No
	Golden Cross Bantam	Yes
	Golden Giant*	Yes
Barley	Gem	Yes
	Dicktoo*	Yes
Wheat	Lemhi	Yes
	Pawnee	Yes
Cane (Sorghum)	Orange	No
	Westland Sorghum*	No
Oats	Overland	No
	Mo-0-205*	Yes
	New Dakold*	No
Rye		
Setaria viridis		Yes
Dactylis glomerata		No
Agrotricum hybrids*	6605	Yes
	51-8899	Yes
	54-721	Yes
	53-6897	Yes
	52-6651-55	No
	52-6782	No

* These species and varieties were tested by and this information is published with the consent of Dr. W. H. Sill, Jr. of Kansas Agricultural Experiment Station, who confirmed the identity of this virus.

symptoms of disease at any time.

CONTROL

Field surveys revealed an unaccountable variation in distribution of wheat streak mosaic in sweet corn-producing areas of the State. The disease appeared to be confined to Golden Cross Bantam and its related inbreds but it was observed that adjacent fields of Golden Cross Bantam often exhibited different reactions. For these reasons a study was conducted to determine the reaction of six different commercial strains of Golden Cross Bantam and related inbreds. The reaction of the F_1 hybrids to manual inoculations were quite variable and the results were inconclusive. The reactions of the inbreds were, on the whole, consistent and predictable. All but one of the male inbreds were either immune or highly resistant but the female inbreds exhibited significant differences in susceptibility. The least susceptible averaged 53.1 percent diseased in three trials while the most susceptible averaged 93.8 percent.

These data strongly suggest that highly resistant strains of Golden Cross Bantam can be obtained by proper selection and mating of inbreds.

IDAHO AGRICULTURAL EXPERIMENT STATION

METHODS OF INOCULATION AND DETERMINING VARIETAL REACTIONS IN THE SEPTORIA DISEASE OF OATS[1]

A. L. Hooker[2]

Abstract

Various methods of inoculating oat plants with Septoria avenae were investigated in the greenhouse and in the field. Type and concentration of inoculum, number of applications, use of wetting and sticking agents, age of cultures, environmental conditions, and other factors were studied in several experiments. Methods of measuring differential varietal reaction to infection in the leaf, stem, glume, and kernel are described.

Septoria avenae Frank is capable of attacking the aerial portions of the oat plant at most stages of its development and, under appropriate environmental conditions, produces characteristic leaf, stem, glume, and kernel infections. Oat varieties and selections differ in their reaction to the pathogen, suggesting that resistant varieties may be a feasible control of the disease. As natural epiphytotics of the Septoria disease may not occur annually, various methods of artificial inoculation and measuring varietal reaction were investigated. This was done so that varietal reactions might be determined each year under conditions favorable for severe disease development and the most resistant oat strains identified. Studies were conducted both in the greenhouse and in the field.

Previous reports have indicated the use of artificial inoculations (1, 2, 3, 4, 5).

INOCULATION METHODS

For the most part, various types of spray or hypodermic inoculations were employed.

Water suspensions of spores, of mycelial segments, or of spores and mycelial segments of the pathogen were applied to plants with an atomizer in the greenhouse or with a knapsack sprayer in the field. Plants were incubated in a moist chamber following greenhouse inoculations. Spray inoculations were used to induce leaf infection on seedlings and adult plants and to induce glume infection in the post-heading stages. Stem infection subsequently developed on spray inoculated plants both in the greenhouse and in the field.

Stem infections also were induced by hypodermic inoculation in the late shoot- or early boot-stage (5). From 1 to 2 cc of inoculum, prepared by macerating 1 Petri plate of culture and substrate in 100 ml of water, was injected into each individual culm. The inoculated culms in a row were identified for subsequent harvest by tying them together with cord or twine. A tag giving date of inoculation, stage of plant growth when inoculated, progeny row number and other pertinent information was attached to the group of inoculated culms.

Septoria infection of the kernel was obtained by hypodermic inoculation in the boot stage. Significant and differential varietal reactions to kernel infection were measured by this method in 1955 and in 1956 (1, 2). Kernel reactions were correlated in successive years (3).

Glume infection was induced by applying inoculum as a spray to plants in the early post-heading stages and incubating the plants in a moist chamber for 36 hours. The plants employed were produced in the field following mid-summer plantings and were transplanted to greenhouse pots before frost in September or October.

Septoria-free adult plants produced in this manner were also used for leaf and stem infection studies under controlled conditions in the greenhouse.

---

[1]Cooperative investigation between the Crops Research Division, United States Department of Agriculture, and the Wisconsin Agricultural Experiment Station. Published with the approval of the Director of the Wisconsin Agricultural Experiment Station.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Madison, Wisconsin. The author is indebted to R. W. Ruhde for technical assistance.

SCORING METHODS

Leaf infection ratings were recorded on a whole plant basis as the percentage of leaf area showing symptoms of the disease. Usually the lower leaves of a plant were more severely infected than the upper. Leaf infection scores ranging from 20 to 75 percent have been measured among oat varieties or selections in artificially inoculated nurseries in the field, and from 5 to 65 percent in greenhouse seedling tests.

Significant differences in size of leaf-lesions also exist among oat strains. These have been recorded on a scale of 1 to 5, with strains expressing small and restricted lesions assigned a score of 1 and those with large and spreading lesions assigned a score of 5.

Maximum leaf infection is usually present approximately 1 week after heading, although ratings may be made earlier or later. Symptom expression, however, may become obscured as plants approach maturity or become infected with other leaf diseases.

Stem infection ratings were taken at maturity. They may be made on plants in the field although smaller differences in varietal reaction can be determined in the laboratory. Ten culms were harvested at random in each plot. The leaves and leaf sheaths were removed and the stem tissue so exposed was placed under uniform lighting conditions and rated as to the percentage of stem area infected. The hypodermically inoculated culms were treated and rated for stem infection in a similar manner. Ratings ranging from a trace to 70 percent infection have been recorded.

Kernel infection ratings were taken on the threshed seed from the hypodermically inoculated culms. These scores were recorded as percentage of infection and took into account both number of kernels infected and degree of infection on individual kernels.

Ratings for glume infection on inoculated panicles were taken 3 weeks after inoculation. They were taken as percentage of infection and employed a scale similar to that for kernel infection.

Disease evaluations were usually replicated and subjected to statistical analysis. This was useful and perhaps necessary as the differences in disease reaction were quantitative. In addition, uniform conditions of inoculation and disease development were difficult to obtain, especially if the number of entries in a test was large.

GREENHOUSE INVESTIGATIONS

Few data were available on techniques of inoculation with Septoria avenae in the greenhouse. Therefore, the effects of spore concentration, length of time in a moist chamber, age of culture, isolates of the pathogen, additions of a wetting agent, and other factors were investigated. A majority of the tests was conducted with seedlings, but inoculations in the shoot, boot and post-heading stages also were successful. All experiments were replicated and involved five or more oat varieties.

S. avenae produces two primary cultural types, i.e. mycelial and pycnidial, on artificial media. The mycelial type is usually obtained when the fungus is first isolated from naturally infected host material, and can be readily established from a pycnidial culture by the transfer of mycelium. This latter method was employed to obtain paired mycelial and pycnidial strains of several isolates of the pathogen. In all comparisons the sporulating strains proved to be more pathogenic than the mycelial strains.

Sporulating cultures and substrate were macerated with water in various proportions in a Waring Blendor. In addition, sporulating cultures were flooded with water and a series of spore concentrations prepared. Higher levels of infection were obtained with macerated cultures and substrate than with spores alone. In a similar manner, inoculum prepared with the culture substrate of mycelial strains of the pathogen was more infectious than that prepared from macerated mycelium alone. The degree of infection was directly proportional to inoculum concentration in all cases. An inoculum concentration of one Petri plate of a sporulating culture and substrate per 100-200 ml water resulted in satisfactory differential infections on the five oat varieties.

In several experiments the degree of infection was directly proportional to the length of time inoculated plants were incubated in a moist chamber, but the increase beyond 36 hours was not significant. Although several schedules were tried, no advantage was gained from alternate wet and dry periods. The results from all tests indicated that abundant and continuous moisture for 36 hours immediately after inoculation was optimum for S. avenae infections in the greenhouse.

Vigorous sporulating cultures usually are obtained in about 7 days when sterile plates of

potato-dextrose agar are flooded with spores of S. avenae. To determine if young or older cultures were most suitable for inoculation purposes, cultures of the same sporulating isolate ranging in age from 4 to 34 days by approximately 2-day intervals were used to inoculate 5 oat varieties in a replicated experiment. Some infection was produced by cultures of all ages but infection was sharply reduced when cultures 16 days old or older were used. Sporulating cultures between 5 and 10 days of age appeared to be the most suitable for inoculation purposes.

The addition of a wetting agent (Tween 20) to the inoculum gave beneficial effects only when the inoculum concentration was low or when agar substrates were not included in the inoculum. There was no apparent gain from its use at inoculum concentrations of one Petri plate of culture and substrate in 100 or 200 ml of water.

S. avenae infections were obtained by placing dry infected leaves with pycnidia on oat seedlings in a moist chamber. Dry leaves collected in the field following heavy natural epiphytotics of the disease also were pulverized mechanically and applied to the seedlings as a dust. More difficulty was experienced in obtaining uniform levels of infection with the dried-leaf dust than with spore and mycelial suspensions prepared from cultures. Therefore, spore suspensions were prepared from infected leaves. The oat leaves containing pycnidia of S. avenae were collected in the field shortly after anthesis, dried in the laboratory for a few days, stored at 12° C, and inoculum prepared by steeping the leaves in water containing a wetting agent. A suspension of 2.5 gm of leaves in 100 ml water containing 2 drops of Tween 20 produced a satisfactory spore concentration for inoculation. Spore concentration, however, would be expected to vary with the extent of pycnidial formation in the infected leaves. The spore suspension was applied as a spray and the inoculated plants then placed in a moist chamber. Abundant and differential infections were obtained.

S. avenae has remained viable in dry infected leaves stored at 12° C for 2 years.

Numerous tests have shown that isolates of S. avenae vary in virulence. Single-spore subcultures from field isolations also differ significantly in virulence. Several isolates and single-spore subcultures can be compared for virulence and the most virulent types used for inoculation either individually or combined.

Cultures of S. avenae maintained in the laboratory on artificial media for 50 or more transfers have shown a reduction in virulence.

Stem infections are believed to be initiated through the secondary spread by water of spores produced in leaf lesions. A test was made to determine if stem infection might be increased through supplementary applications of water after leaf lesions were well developed. In this evaluation adult plants of 30 oat varieties in a replicated experiment were inoculated in a moist chamber. All plants were incubated in a common greenhouse for 2 weeks following inoculation and abundant leaf infection was obtained. Two replicates of the 30 varieties were moved to a second greenhouse, where an overhead spray maintained a high humidity and the plants were wetted frequently. The remaining plants were kept in a dry atmosphere. Three weeks later all plants were moved to a dry atmosphere and allowed to ripen before stem infection ratings were taken. The average stem infection for the 30 oat varieties exposed to high humidity was 37.7 percent and for those maintained at low humidity was 27.6 percent. This difference was highly significant statistically.

FIELD INVESTIGATIONS

The relative efficiency of various methods of inducing artificial epiphytotics of the Septoria disease was studied in the field.

Six replications of 15 plots, each plot consisting of a 3-foot row of each of five oat varieties (Andrew, Branch, Fayette, Rodney, and Sauk) in random order and bordered by a row of Ajax, were sown on April 23, 1955. The inoculation treatments listed in Table 1 were assigned to each plot at random. Treatments on June 8 were scheduled for 3 P.M. and 7 P.M. but were made at 4 P.M. because of weather conditions. Spray treatments consisted of a mixture of sporulating cultures that had proved to be highly pathogenic in greenhouse tests. Cultures were macerated in a Waring Blendor in the proportion of 1 Petri plate of a 10- or 12-day-old culture per 100 ml water and this concentrate was diluted 1:5 with water for field application. Gelatine and Tween 20 were added as indicated in Table 1. The oat leaf dust consisted of pulverized leaves collected from heavily infected plants the previous season.

The May 25 inoculations were made on a cool cloudy day following a 0.83 inch rainfall the previous day and night. The night of May 25-26 was cold and heavy dews were formed. June 1 was also a cloudy day following rain. A light mist occurred at 7:30 P.M. followed by a cool night. Intermittent showers and cloudy weather prevailed on June 2. Rain occurred in the morning of June 8 as well as during the previous 4 days, and the plants were wet when sprayed

Table 1. Average leaf and stem infection with Septoria avenae on five oat varieties (Andrew, Branch, Fayette, Rodney, and Sauk) following various methods of inoculation at Madison, Wisconsin in 1955 and a summary of the analyses of variance. Leaf readings are the average of 6 replications and stem readings are the average of 5 replications.

Inoculation Method	% Leaf infection on 6/22	6/29	% Stem infection
Uninoculated check	20.2	29.6	29.8
Spray[a] on May 25, June 1, 8, 16, & 20; 7 P.M.	35.4	40.3	27.2
Spray on May 25, June 8 & 20; 7 P.M.	34.5	35.5	29.2
Spray on June 1 & 16; 7 P.M.	34.0	34.0	30.4
Spray on June 8 & 20; 7 P.M.	36.0	37.8	33.0
Spray on June 16; 7 P.M.	24.3	30.8	34.6
Spray on June 8; 4 P.M.	35.7	40.9	29.8
Spray on June 8; 4 P.M.	36.1	38.0	29.6
Spray + .25% gelatine on June 8; 4 P.M.	34.2	40.2	28.2
Spray + .25% gelatine on June 8; 4 P.M.	34.7	40.5	34.4
Spray + Tween 20 on June 8; 4 P.M.	41.2	45.2	37.0
Spray + Tween 20 on June 8; 4 P.M.	43.2	45.6	34.2
Oat leaf dust[b] on June 8; 4 P.M.	26.7	31.2	35.0
Oat leaf dust on June 8; 4 P.M.	30.5	30.2	35.0
Water spray + oat leaf dust on June 8; 4 P.M.	31.5	32.0	37.2
L.S.D. .05	13.6	9.2	12.1
L.S.D. .01	18.1	12.3	16.1

Summary of Analyses of Variance

Source of Variation	d.f.	S.S.	M.S.	F.
Leaf infection on 6/22	14	74,526.67	5,323.33	7.58**
Leaf infection on 6/29	14	240,340.56	17,167.18	26.71**
Stem infection	14	18,788.67	1,342.05	2.95**

** Significant at 1% level

[a]Spray inoculum was a composite of sporulating cultures macerated in a Waring Blendor and mixed with water in the proportion of one Petri plate of culture to 600 ml of water.

[b]Dry pulverized leaves collected from diseased plants the previous season.

or dusted. Light showers occurred during the night and following morning. The June 16 sprays were applied on a clear day. Dew formed on the plants at 8:00 P.M. and persisted until 7:45 A.M. on June 17. June 17 was warm and clear. The June 20 inoculations were made on a hot, dry day and only a moderate dew formed on the plants overnight. June 21 was partly cloudy and warm.

The average leaf infections on a whole plant basis recorded on June 22 and 29 are presented in Table 1. All inoculation treatments resulted in an increased infection over that of the check. Spray inoculations made on June 8 generally resulted in the highest incidence of leaf infection. This may have been due in part to weather conditions and in part to stage of plant growth. The data indicate that the addition of a wetting agent such as Tween 20 may increase the infection, but no gain was apparent from the addition of gelatine to the inoculum.

Percentage of stem area infected on each entry was taken at maturity on 2 replicates in the field and on 3 replicates in the laboratory. The leaf and leaf sheaths were removed from 10 culms of each sample in the laboratory to facilitate the observation of disease development. The average stem infection on the 5 varieties in 5 replications for each method of inoculation is presented in Table 1. Although significant differences were measured among inoculation treatments, they were not so large as those for leaf infection. Considerable variability among replications and within treatments was evident. This may have been due in part to the severe and differential lodging which occurred in the test 4 weeks prior to maturity.

The tendency toward uniformity of stem infection on a given oat variety in different plots, despite different degrees of leaf infection, was also illustrated in other tests in which numerous sets of check varieties inserted in a variety evaluation test received a spray inoculation. In these latter tests significant differences were measured among the sets of check varieties in leaf infection while differences in stem infection were non-significant. The differences in leaf infection were presumably due to the inability to apply a constant amount of inoculum over a large area.

Field plots seeded on April 19, 1956 were inoculated by spraying on May 31 and June 7, 11, and 14. The inoculum was applied in the late afternoon when the humidity was high or just before sunset on bright days. The inoculum consisted of a composite of sporulating and highly pathogenic cultures. Cultures in approximately 400 Petri plates were blended in a Waring Blendor and mixed with 70 gallons of water for each application. A wetting agent (Tween 20) was added to the inoculum to reduce surface tension. Numerous well developed lesions were obtained following each inoculation. Although the weather was mostly warm and dry throughout the inoculation period, 75 percent of the leaf area on susceptible varieties became infected. Abundant stem infection developed subsequently on susceptible oat strains in the inoculated plots. Statistically significant varietal differences in leaf and in stem infection were measured in the inoculated plots despite the low incidence of disease in adjacent plots exposed only to natural infection.

SUMMARY AND DISCUSSION

Significant differences among oat varieties and selections in reaction to Septoria avenae in the leaf, stem, glume, and kernel can be measured with artificial inoculations in the field or in the greenhouse. Oat varieties or selections have reacted in a similar but differential manner to all methods of artificial inoculation and to natural infection (3).

The inoculation technique found most suitable for use in the greenhouse consists of blending 7- to 9-day-old, sporulating, pathogenic cultures on potato-dextrose agar with water in a Waring Blendor. These are blended for 1 to 2 minutes at the rate of 1 Petri plate of culture and substrate per 100 ml of water. The suspension is passed through one thickness of cheese cloth and sprayed on the plants with an atomizer at 15 pounds pressure. Inoculated plants are incubated in a moist chamber for 36 hours. Lesions appear in 3 days and notes can be taken after 7 to 9 days. These inoculations can be made on plants of any size and have been successfully used to measure varietal reactions in the host and variations in virulence in the pathogen.

For field spray applications, the inoculum is diluted 1:5 with water and a wetting agent such as Tween 20 added to reduce surface tension. One Petri plate of culture will provide sufficient inoculum for 6 to 10 3-foot nursery rows. Three to 5 spray inoculations are made between 6 and 8 weeks after seeding. They are usually made in the late afternoon or early evening depending upon atmospheric humidity. Greater infections are likely to result if field inoculations are made when there is free moisture on the foliage.

Satisfactory leaf ratings have been obtained by recording leaf infection as a percentage of

the leaf area expressing lesions, on a whole plant basis. Varietal differences in lesion size are correlated with total leaf infection (3) and appear to be an expression of resistance.

Late oat varieties frequently had a lower leaf infection rating at late dates of reading than at an earlier date. This may be due in part to disease escape, as many of the upper leaves on late varieties do not emerge early enough to be exposed to inoculum or to suitable environmental conditions for infection. Subsequent stem infections were also associated with late maturity (3)

Stem ratings, based upon the percentage of stem area of a whole plant expressing symptoms of infection, preferably are made after removing the leaf sheaths. Although the ratings on stripped and unstripped stems are correlated ($r = 0.596^{**}$ for 170 paired comparisons), a greater range in infection and perhaps a more accurate rating for stem infection can be recorded if the leaf sheath tissue is first removed. Hypodermic inoculations increase the amount of local infection, are more variable, and frequently result in considerable plant injury. They may have value, however, under conditions in which stem infection does not follow spray inoculations or natural infection.

Glume, leaf, and stem infection can be obtained on adult plants, transplanted from the field to pots and spray-inoculated in a greenhouse moist chamber. This technique is useful in fundamental studies of the pathogen or of the disease.

Kernel infection ratings at maturity following hypodermic inoculations in the boot stage revealed varietal differences but with high variability. Varietal reactions, however, were similar in consecutive years (3).

Most of the artificial inoculations have employed inoculum produced by growing the pathogen in culture, but spore suspensions obtained by soaking infected oat leaves containing pycnidia in water were used successfully in some greenhouse inoculations. Presumably inoculum so prepared also would be effective for use in the field. The addition of a wetting agent to the water appears to facilitate the release of spores from the pycnidia and coverage of inoculated plants.

Differences in varietal reaction to the Septoria disease in oats are quantitative and influenced by environmental conditions. Therefore, disease evaluations are usually replicated and subjected to statistical analysis.

Literature Cited

1. HOOKER, A. L. 1955. Septoria reactions of entries in the North Central States uniform oat yield nursery and other strains in 1955. Plant Dis. Reptr. 39: 963-966.
2. HOOKER, A. L. 1957. Septoria reactions of oat varieties and selections in 1956. Plant Dis. Reptr. 41: 385-388.
3. HOOKER, A. L. 1957. Varietal reaction correlation studies in the Septoria disease of oats. Agron. Jour. 49: In press.
4. HUFFMAN, M. D. 1955. Testing for resistance to the Septoria disease of oats. Plant Dis. Reptr. 39: 25-28.
5. LUND, STEVE, and H. L. SHANDS. 1956. The response of oat strains to Septoria avenae. Phytopathology 46: 48-50.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, MADISON, WISCONSIN

RATHAY'S DISEASE OF ORCHARD GRASS FOUND IN VIRGINIA

A. S. Williams and L. H. Taylor[1]

Rathay's disease of orchard grass, Dactylis glomerata L., caused by Corynebacterium rathayi (E. F. Smith) Dowson, was identified from specimens of Virginia commercial orchard grass collected near Upperville, Virginia, May 22, 1957. This disease was previously reported from Oregon in 1945[2].

The symptoms are characteristic of those described by E. F. Smith[3] and are illustrated in Figure 1. A yellow bacterial slime (black in photograph) appears on the upper surface of the plant causing a dwarfing and distortion of the uppermost leaves, the upper part of the stem and parts of the inflorescence. Because of the sticky slime, the stems often form knee-shaped bendings (see photograph) at the uppermost internode due to continued elongation of the basal portion of the stem when the inflorescence is cemented to the leaf sheath.

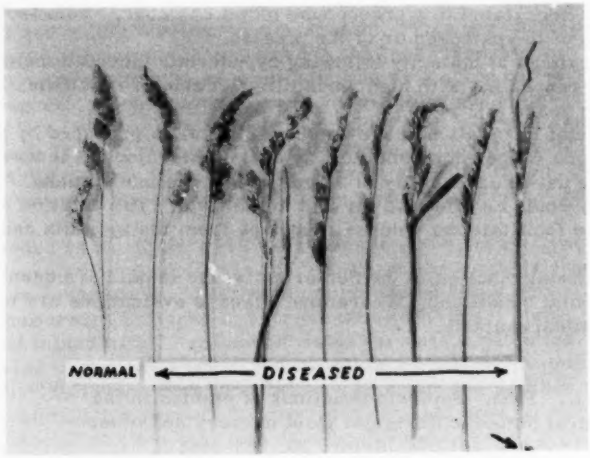


FIGURE 1. Rathay's disease on inflorescence of orchard grass, showing increasing amounts of disease from left to right. Arrow (lower right) indicates typical "knee-bend".

A survey made for this disease May 29 and 30 covered portions of eight counties in the major seed producing area. These counties with the number of fields examined were: Fauquier (30), Loudoun (19), Prince William (2), Clarke (10), Shenandoah (5), Frederick (3), Rockingham (3), and Augusta (3). Severe infestations were observed at two locations; one in Fauquier and one in Loudoun County. Trace amounts of disease were observed at one additional location in Fauquier, two in Loudoun, and one in Prince William County. A more extensive survey is needed to determine its distribution in Virginia.

VIRGINIA AGRICULTURAL EXPERIMENT STATION

---

[1]Associate Plant Pathologist and Agronomist, respectively, Virginia Agricultural Experiment Station, Blacksburg, Virginia.

[2]Hardison, J. R. 1945. Bacterial blight of orchard grass observed in Oregon. Plant Dis. Reptr. 29: 600.

[3]Smith, E. F. 1914. Bacteria in Relation to Plant Disease III. Carnegie Institute of Washington, Washington, D. C.

RADOPHOLUS GRACILIS IN A DRY SUBHUMID ENVIRONMENT

Don C. Norton

Radopholus gracilis (de Man, 1880) Hirschmann, 1955, has been reported to occur in extremely moist places such as bogs, marshes, lakes, and river banks. Many of the plants with which the nematode has been associated are hydrophytes, and those not classed as such were growing in extremely mesic conditions such as listed above. These habitats are summarized by Hirschmann (4). Golden (2) recently found R. gracilis associated with reeds and bulrushes along a river bank in California.

As Hirschmann (4) has pointed out, R. gracilis and R. oryzae (v. Breda de Haan, 1902) Thorne, 1949, may be synonymous, and in one publication (5) he actually listed them as such. R. oryzae is known to parasitize rice in Java and Japan. It has also been found in rice fields in Texas (1). Hirschmann (3) found it around the roots of the marsh marigold, Caltha palustris L., from the edge of a brook in Germany.

Radopholus gracilis was found in September, 1956, associated with roots of Virginia wild rye, Elymus virginicus var. glabriflorus (Vasey) Bush growing in a clay loam upland slope in the rolling plains of Archer County, Texas. This area has a dry subhumid climate with an average annual rainfall of 23.5 inches and a precipitation/evaporation ratio of approximately 0.53. Much of the moisture falls as local showers and torrential rains resulting in much runoff. From 1951 to 1956 inclusive has occurred the most severe drouth on record. An average population of 1160 of R. gracilis per pint of soil was obtained.

Since the present finding is in a distinctly more xeric habitat than previously reported, it was thought to be of interest. It was found, however, only in one location during a general survey of nematodes on small grains and native grasses in north Texas.

Literature Cited

1. ATKINS, J. G., M. J. FIELDING, and J. P. HOLLIS. 1955. A new nematode on rice in Texas and Louisiana. Plant Dis. Reptr. 39: 69.
2. GOLDEN, A. M. 1957. Occurrence of Radopholus gracilis (Nematoda: Tylenchidae) in the United States. Plant Dis. Reptr. 41: 91.
3. Hirschmann, H. 1954. Unerwarteter Wiederfund tropischer Nematoden (Radopholus oryzae v. Breda de Haan, 1902) Thorne, 1949, Panagrolaimus hygrophilus Bassen, 1940, Atylenchus decalineatus Cobb, 1913 an heimischen Sumpfpflanzen. Zeitschr. f. Pflanzenkrankheiten (Pflanzenpathologie) u. Pflanzenschutz. 61: 352-357.
4. HIRSCHMANN, H. 1955. Radopholus gracilis (De Man, 1880) n. comb. (Synonym-Tylenchorhynchus gracilis (De Man, 1880) Filipjev, 1936). Proc. Helm. Soc. Wash. 22: 57-63.
5. HIRSCHMANN, H. 1955. Tylenchorhynchus gracilis (De Man, 1880) Filipjev, 1936-Radopholus gracilis (De Man, 1880) n.c. und seine Synonyme. Zoologischer Anzeiger. 154: 288-301.

DEPARTMENT OF PLANT PHYSIOLOGY AND PATHOLOGY
TEXAS AGRICULTURAL EXPERIMENT STATION
COLLEGE STATION, TEXAS

RECOMMENDED CHANGES IN RECOVERY TECHNIQUES FOR BURROWING NEMATODE

J. A. West[1]

The moist chamber root incubation technique developed by T. W. Young[2] is one of the most practical methods for recovery of migratory endo-parasitic nematodes such as Radopholus similis. In adapting this technique for use in survey work by the Florida State Plant Board and the Federal Plant Pest Control Division, an attempt was made to substitute one examination for the repeated syringings and examinations of the Young method. While the moist chamber technique has worked quite well under these conditions, several factors were noted which led to this investigation.

The standard incubation period has generally been 3 to 5 days. This period was determined by the reasonable numbers of burrowing nematodes recovered and the necessity of preventing the build-up of conditions unfavorable to the nematodes during a longer incubation period. However, it was recognized that an additional period of incubation after flushing the roots with fresh water very likely would recover additional nematodes from the roots.

During the course of a four-day moist chamber incubation period, it was noted that roots in the top part of the jar became dry. Since burrowing nematodes need a film of moisture for survival, it was unlikely that the maximum number of nematodes would be recovered from the roots in the upper section of the jar. Covering the entire root ball with water offered a possible solution to this problem.

The first investigations were directed toward determining the possible beneficial effects of completely immersing the roots in water and the resulting detrimental effects of reducing the available oxygen supply. A series of samples incubated in a chamber where the oxygen was replaced by carbon dioxide decisively showed that the number of burrowing nematodes recovered was reduced and nearly all of the nematodes were dead.

Other series of samples were incubated in water and compared with a check series of moist chamber incubations for periods of 2 days and 7 days. A considerable increase in the number of burrowing nematodes recovered was noted in the water-incubated samples. There was no difference in the condition of the nematodes at the end of a two-day period; however, it was noted after 7 days of uninterrupted incubation that most of the nematodes were in poor condition or dead where roots were completely immersed in water, while nearly half the nematodes were still alive in the moist-chamber samples.

Results of these preliminary studies led to further investigation to determine actually how rapidly burrowing nematodes are recovered. A series of three samples was processed with half the samples incubated in a moist air chamber and the other half incubated in water. This study was replicated on three separate dates. Readings were taken each day for 5 days and then again on the eighth day.

A grand total of 5,208 burrowing nematodes was recovered during the eight-day period. Total numbers of live nematodes recovered were nearly equal in the two methods. There was an apparent beneficial effect to all samples from flushing the roots with fresh water each day. The one great difference between water immersion and moist air chamber incubation is the rate of emergence of the endo-parasitic nematodes. Eighty-one percent of the burrowing nematodes emerging during an eight-day period was recovered in one day by the water method. A period of 4 to 5 days was required to recover equal numbers from the moist chamber.

CONCLUSIONS

The water incubation method offers many possibilities for increased recovery of burrowing nematodes from citrus roots. More reliable results can be obtained with a single examination of a sample when the roots are incubated in water. The incubation period can be reduced by several days. Time saved in the incubation period will permit much quicker completion of the delimiting surveys, since repeated inspections must be made to determine the fringes of the infestation.

There are some minor disadvantages in the use of the water incubation method. Samples cannot be held as long before examination due to the earlier death of the nematodes and their

[1]Laboratory Supervisor, Southern Region, Plant Pest Control Division, United States Department of Agriculture, Lake Alfred, Florida.

[2]Young, T. W. 1954. An incubation method for collecting migratory endo-parasitic nematodes. Plant Dis. Reptr. 38: 794-795.

Table 1. Numbers of burrowing nematodes recovered from citrus roots in periodical examinations during eight days incubation.

Replication* (Beginning Date of Incubation)	Treatment	First 24 hrs.	Second 24 hrs.	Third 24 hrs.	Fourth 24 hrs.	Fifth 24 hrs.	Sixth, Seventh & Eighth 24 hrs.	Total
3/31/57	Moist Air Chamber	266	130	96	98	106	104	800
	Water-Filled Chamber	526	38	8	4	8	38	622
4/7/57	Moist Air Chamber	406	156	156	132	85	216	1151
	Water-Filled Chamber	776	106	23	13	7	80	1005
4/21/57	Moist Air Chamber	338	144	74	48	39	90	733
	Water-Filled Chamber	742	60	15	37	22	21	897
Totals of Three Replications	Moist Air Chamber	1010	430	326	278	230	410	2684
	Water-Filled Chamber	2044	204	46	54	37	139	2524
							Grand Total	5208
Totals Expressed in Percentages	Moist Air Chamber	37.63	16.02	12.15	10.36	8.57	15.28	
	Water-Filled Chamber	80.98	8.08	1.82	2.14	1.47	5.51	

* Each replication consisted of three individual samples divided equally between the two treatments.

more rapid deterioration. Field and laboratory work, therefore, must be kept in much closer balance. The increased weight of samples and greater difficulty in preventing leakage from jars in transportation may prohibit use of the water method with samples collected in field stations removed from the laboratory where examination will be made.

REMARKS

Samples collected at the Lake Alfred station were incubated in water beginning May 1, 1957. The method will be given critical evaluation to determine whether it can be adapted for practical use with all root samples taken in connection with the burrowing nematode program.

SOUTHERN REGION, PLANT PEST CONTROL DIVISION, UNITED STATES DEPARTMENT OF AGRICULTURE, LAKE ALFRED, FLORIDA

PATHOGENICITY TESTS INVOLVING MELOIDODERA FLORIDENSIS, A NEMATODE ASSOCIATED WITH SLASH PINE

B. G. Chitwood and R. P. Esser[1]

Summary

Meloidodera floridensis reproduced and increased itself on Pinus elliottii seedlings without apparent injury to the roots or tops. Males were obtained from root incubations at a ratio of about 1000 larvae to 1 male.

On November 9, 1955 slash pine seedlings were collected from the United States Forest Service Nursery at Olustee, Florida. The purpose of these collections was to obtain specimens of the new nematode genus Meloidodera for anatomic study and to initiate pathogenicity tests. The following are results of the preliminary pathogenicity experiments.

METHOD AND PROCEDURE

Twenty slash pine seedlings were removed from a sterile seedbed and replanted in 3-inch pots. Two gravid Meloidodera floridensis females were placed adjacent to the roots in each of ten potted seedlings. The ten remaining pots served as checks.

Two months after inoculating, one of the inoculated seedlings and its check were examined with negative results. Six months later, July 7, 1956, the remainder of the seedlings were examined.

Roots were cut at the soil line, washed, weighed, and then examined on both sides under a binocular microscope and protruding females were counted. The soil from each pot was Baermannized and the larva collected from each Bearmann counted.

Larval counts were also made from the washed roots after incubating them for three days at room temperature.

RESULTS

Results of these examinations are presented in Table 1.

DISCUSSION

In the present experiment it would appear that there was a minor, nonsignificant, increase in root growth as a result of the light inoculation with Meloidodera floridensis. On the basis of prior experimental work with Meloidogyne spp.[2] such stimulation of plant growth with mild infections is to be expected. We may also expect reduction in root growth as well as top growth with more massive inoculations.

Experiments are in progress to determine whether or not symptoms will be produced in Pinus elliottii with a substantial increase of inoculum.

---

[1]Chief Nematologist and Assistant Phytonematologist, respectively, Florida State Plant Board, Gainesville, Florida.

[2]Chitwood, B. G., A. W. Specht, and L. Havis. 1952. Effects of Meloidogyne incognita and M. javanica on some peach rootstocks. Plant and Soil 4: 77-95.

Table 1. Results of inoculations with Meloidodera floridensis on slash pine (9 plants)

Inoculation Number	Mature Females	Larva In Soil	Larva from Roots (3 days)	Males	Root Weight (grams)
(1)	29	340	291	0	6.4
(2)	75	1073	1007	2	2.6
(3)	62	353	647	0	5.1
(4)	0	0	0	0	3.3
(5)	16	1	23	0	2.6
(6)	9	17	204	0	5.8
(7)	58	317	638	1	6.5
(8)	56	372	208	0	2.8
(9)	8	204	212	0	6.9
Total	313	2677	3230	3.0	42.0
Mean	34.7	297.4	358.8	.3	4.7
Control					
(1)	0	0	0	0	7.0
(2)	0	0	0	0	4.7
(3)	0	0	0	0	4.6
(4)	0	0	0	0	4.0
(5)	0	0	0	0	3.6
(6)	0	0	0	0	3.7
(7)	0	0	0	0	5.2
(8)	0	0	0	0	5.6
(9)	0	0	0	0	No roots
Total	0	0	0	0	38.4
Mean	0	0	0	0	4.2

FLORIDA STATE PLANT BOARD

EFFECT OF IRRIGATION FREQUENCY AND SIZE OF TOMATO SEEDLINGS ON ROOT KNOT INDEX

Bakir A. Oteifa and Dawood M. El-Gindi

Summary

The effect of irrigation frequency and size of tomato seedlings on disease index were determined under field conditions. Results proved that plants were severely injured under heavy irrigation treatments. On the other hand, the effect of seedling size did not account for significant results.

PROCEDURE

In connection with an experiment carried out in the Department of Horticulture of Cairo University, Giza, Egypt, for studying the effect of irrigation frequency and seedling size on the growth and yield of tomatoes, it was suggested that the effects of these two factors on the degree of nematode infection should be determined.

Seedlings of tomatoes Var. 13 were propagated in wooden flats. After 2 months, seedlings were pulled out and classified according to their size as: large, approximately 12 inches long; medium, approximately 8 inches long; and small, approximately 5 inches long. These seedlings were transplanted to a field plot known to be heavily infested with root-knot nematodes (Meloidogyne sp.). This field plot was designed according to the split plot plan. Four replicates were used and each was divided into three plots. The plots were irrigated as follows: one plot once a week, the second plot once every other week, and the third plot once every three weeks. During the growing season, all the plots were horticulturally treated alike.

After the plants had reached maturity, roots were pulled gently and rinsed in tap water. Root systems were examined visually and indexed according to a modification system used by Smith and Taylor (1) to the following categories: 0 = no evidence of galling; 1 = small gall formation; 2 = moderate galling; and 3 = extensive galling. The disease index of each plot was calculated according to the following formula:

$$\text{Disease index} = \frac{\Sigma \text{ Category numbers}}{\text{No. of plants} \times 3} \times 100$$

The category numbers were obtained by multiplying the number of plants in each category by their respective category number. Analysis of variance was applied to the data according to the method of Snedecor (2).

RESULTS

The analysis of variance given in Table 1 reveals that the different irrigation treatments exerted a significant effect on the disease index at the 5% level of probability. On the other hand, the different sizes of seedlings used did not account for such significant effect. Their interaction also did not exert any significant difference. This indicates that the irrigation treatments were considered as the main important factor determining the degree of disease index under the conditions of this experiment. For this reason, a comparison analysis as shown in Table 2 was necessary to evaluate the degree of irrigation frequency for the different treatments. This indicates that plots irrigated every week had a non-significant effect at the 1% level when compared with plots irrigated every other week. However, when comparison was made between plots treated every week and plots treated every 3 weeks, results were significant at the 1% level of probability. The comparison between irrigated plots treated every other week and every 3 weeks was also highly significant at the 1% level, indicating that root-knot nematodes were more active in heavily irrigated plots and consequently that plants were severely injured. Table 1 also shows a non-significant variability between the three sizes of seedlings at the 5% level of probability.

Table 1. Analysis of variance of the effect of irrigation frequency and size of tomato seedlings on root knot index.

Source of variation	Degree of freedom	Sum of squares	Variance	F value
Main plots:				
Irrigation treatment	2	1278.9	639.48	5.48*
Blocks	3	316.0	105.33	0.91
Error	6	699.35	116.55	--
Sub plots:				
Seedling size treatment	2	352.6	176.3	2.91
Seedling size irrigation	4	53.0	12.5	0.20
Error	18	1090.4	60.58	--
Total	35	3790.25	--	--

* Significant at the 5% point.

Table 2. Comparative analysis of root knot index of tomato plants between irrigation treatments and seedling size treatments.

Source of variation	Degree of freedom	Sum of squares	Variance	F value
Irrigation treatments*:				
A vers B	1	41.34	41.34	0.86
A vers C	1	1092.70	1092.70	22.90**
B vers C	1	742.60	742.60	15.56**
Seedling size treatments*:				
S vers M	1	121.5	121.5	2.54
S vers L	1	348.8	348.8	7.31
M vers L	1	58.6	58.6	1.22
Error	29	1384.9	47.7	-
Total	35	3790.25	-	-

(*)A -- irrigation every week
B -- irrigation every other week
C -- irrigation every 3 weeks
S -- small seedlings
M -- medium seedlings
L -- large seedlings
(**) Significant at the 1% point.

CONCLUSIONS

The rate of disease development is largely connected with the conditions prevailing in the field. In this connection, a question was raised as to whether seedling size and frequency of

irrigation treatments might affect the course of disease development. The present work has shown that seedling size was of minor significance whereas irrigation was an important factor in determining the degree of disease manifestation. Heavy irrigation increased the amount of nematode injury. An explanation of this phenomenon may be that eggs are capable of remaining almost dormant under dry conditions while moisture stimulates them to hatch and infest the field with a fresh and vigorous population of larvae. The infectious hatching larvae are very active in wetter soils. Films of irrigation water also carry nematode larvae from one plant to another, thereby serving as a means of dissemination to all growing plants.

Literature Cited

1. SMITH, A. L., and A. L. TAYLOR. 1947. Field method of testing for root-knot infestation. Phytopathology 37: 85-93.
2. SNEDECOR, G. W. 1946. Statistical Methods Applied to Experiments in Agriculture and Biology. 4th ed. Iowa State College Press. pp 485.

PLANT PROTECTION DEPARTMENT, FACULTY OF AGRICULTURE, CAIRO UNIVERSITY, GIZA, EGYPT

BLUE SPOTTING OF POTATO TUBERS IN BRITISH COLUMBIA[1]

N. S. Wright[2]

Abstract

A physiological disease of potato tubers known in Holland as blue spotting has been identified in late crop tubers in British Columbia. Bluish black discolorations in cortical and pith tissue are incited by handling bruises and they occur only in tubers predisposed to the disease by some adverse environmental factor. In British Columbia blue spotting was most prevalent in tubers grown on farms where soil moisture was inadequate during the latter part of the 1956 growing season.

An internal bluish-black spotting was a serious grade defect in the 1956 late crop tubers (principally Netted Gem) from a number of farms in the Lower Fraser Valley of British Columbia and in certain lots imported from the State of Washington. The same disorder has been observed in previous years in both local and imported lots but was more prevalent in the 1956 crop than in any previous crop on record. The bluish-black spots or patches occur in cortical and pith tissue and are most prevalent at the stem end of the tuber. A bluish-black sheen may be seen on the skin of washed tubers directly over affected tissue and frequently adjacent to a visible injury. These symptoms are shown in Figure 1.

When affected tubers are cooked the discolored areas become dark gray or black and seriously detract from the appearance of products such as potato chips (Fig. 2).

Although the cause of this defect is not known with certainty, there is evidence to indicate that the cause is physiological. Wound cork does not develop around discolored tissue, attempts to isolate a causal organism have failed, and the disease was readily induced in susceptible tubers by bruising. Bruises inflicted by means of a dull instrument in such a way that the skin was not broken or by dropping tubers from a height of 2 feet onto a wooden bench caused the disease to develop within 24 hours in lots known to be susceptible to the disease. Tubers from lots in which the disease had not been previously found failed to develop the disease after bruising.

Published records indicate that an identical or very similar disease has occurred periodically in Great Britain, Holland, and eastern United States. A similar disease considered to be physiological in origin was described in England by Horne (7) in 1913. His description and illustrations of the disease known as "bruise" correspond in every detail with the disease described herein. Several reports from Holland (1, 3, 4, 6, 8, 11) published during the period 1927-1936 describe a disease known as blue spotting which also appears to be identical to the one affecting potato tubers in the Pacific Northwest. The name "blue spotting" is adequately descriptive of the disease and has been applied to the local disease in preference to "bruise" and to "internal blackening" or "internal black spot" which have been used in more recent English (5) and American (12) references.

Blue spotting in Holland was shown to occur most extensively in crops grown in potash-deficient soils and the disease was greatly reduced by application of 40 percent potash salts at the rate of 350 to 700 pounds per acre. The Dutch workers agree, however, that potash deficiency merely renders the tubers susceptible to injuries from rough handling which results in the discoloration. The presence of oxygen was found by Botjes (3) to be essential to the production of the blue color. Mulder (10) concluded that lack of potash had a greater effect than lack of nitrogen, phosphate, magnesium, or copper on increasing tyrosine content of potato tubers and that the two factors responsible for the tendency for tissues to darken are high tyrosine content and liability of the cells to sustain injury. The tendency for blue spotting to occur more commonly in stem-end tissue than in distal end tissue was investigated by Biewenga (1) who found that there was insufficient difference in potash content between

---

[1]Contribution No. 1613 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2]Plant Pathologist, Plant Pathology Laboratory, Science Service, Canada Department of Agriculture, Vancouver, British Columbia.

FIGURE 1. Blue spotting in a Netted Gem potato tuber. Left, external bluish-black sheen near a visible injury; center, surface peeled to show blue spotting; Right, same tuber cut deeper to show extent of blue spotting.

FIGURE 2. Potato chips made from tubers with blue spotting showing undesirable black blotches.

tissue from the two ends to account for the difference in susceptibility. Differences in specific gravity were correlated by de Bruyn (6) with differences in susceptibility to bruising and consequently to blue spotting. Tuber lots with high specific gravity were shown to be more susceptible to bruising (and to blue spotting) than tuber lots with lower specific gravity. She also showed that tissue from the stem end of tubers had a higher specific gravity than tissue from the distal end. Varietal differences in susceptibility were noted by Botjes (3) and by van der Waal (11). Soft-fleshed varieties were observed to be more liable to blue spotting than hard-fleshed ones.

McIntosh (9) showed that internal discoloration of tubers in Scotland was associated with bruising and was most extensive in tubers in which the bruising did not cause surface cracks. Boyd (5) in England also found that bruising incited internal darkening of tubers. He related the extent of internal darkening to the degree of flaccidity of the tissues and showed that susceptibility to bruising increased with the degree of maturation of the tuber.

In the United States, a disease called internal black spot (12) was serious in Long Island potatoes during the 1943-44 storage season. The description of this disease indicates that it was probably the same as blue spotting. Discoloration was produced experimentally by bruising. Tubers were especially susceptible at "pressure bruises", the points of contact between tubers in a pile.

Although the association of handling bruises and blue spotting has been established experimentally, the chemical reactions involved in the discoloration and the conditions which predispose tubers to blue spotting are not completely understood. However, Botjes' (3) demonstration that oxygen was essential to the production of the blue color and Mulder's (10) demonstration that a deficiency of potash increased the tyrosine content of tubers strongly suggests that an enzymic reaction is involved. The potato tuber is known to be a rich source of the enzyme polyphenoloxidase, which is present in the cytoplasm as a constituent of the soluble cytoplasmic protein. According to Bonner (2) "the enzyme has as its substrate monophenols such as p-cresol and tyrosine, oxidizing them to the corresponding o-diphenols and for this reason has also been called tyrosinase. Polyphenoloxidase is, however, in addition capable of catalyzing the oxidation of the ortho-diphenols to the corresponding ortho-quinones. The enzyme is strictly aerobic and the oxidations are carried out only in the presence of oxygen. The quinones produced show a great tendency toward further oxidation and polymerization with the production of colored products, the exact color depending on the phenol used as substrate."

The conditions which in Holland predisposed potato tubers to blue spotting following handling bruises were identified as potash deficiency (3) and high specific gravity (6). In England, loss of turgidity (5) is considered to be the major predisposing factor while in Scotland (9) and United States (12) handling bruises alone are emphasized. In British Columbia, the occurrence of blue spotting could not be correlated with soil type as it occurred equally in tubers grown on clay, clay loam and peat. The disease occurred in crops that had received ample quantities (up to 20 tons per acre) of barnyard manure and in crops that received recommended amounts of complete fertilizer. The only indication that nutritional factors were involved came from the observation that crops which suffered from a shortage of moisture late in the growing season were most severely affected with blue spotting.

Literature Cited

1. BIEWENGA, E. 1929. Potash content and the "blue spotting" of the potato (Trans. title). De Veldbode, Maastricht 1: 382 and 676. (Abst.). R.A.M. 9: 54.
2. BONNER, J. 1950. Plant Biochemistry. Academic Press, Inc., New York. pp. 181-182.
3. BOTJES, J. O. 1927. The blue discoloration of potatoes (Trans. title). Tijdschr. over Plantenziekten 33: 57-96. (Abst.) R.A.M. 6: 632-633.
4. BOTJES, J. O. 1929. Notes on the connection between "blueing" of potatoes and potash shortage (Trans. title). Tijdschr. over Plantenziekten 35: 5-8. (Abst.) R.A.M. 8: 459.
5. BOYD, A. E. W. 1951. The internal blackening of potatoes caused by bruising. Jour. Hort. Sci. 26: 148-156.

6. de BRUYN, H. L. G. 1929. The blue discoloration of potatoes (Trans. title). Tijdschr. over Plantenziekten 35: 186-222. (Abst.) R.A.M. 9: 53-54.
7. HORNE, A. S. 1913. Contributions from the Wisley Laboratory. XVI. Bruise in potato. Jour. Roy. Hort. Soc. 38: 40-50.
8. HÜLSEN. 1936. Black spotting (blue cooking) in potatoes (Trans. title). Ernähr. Pfl. 33: 55. (Abst.) R.A.M. 15: 526.
9. McINTOSH, T. P. 1935. Discoloration of potatoes. Scottish Farmer, 1st June.
10. MULDER, E. G. 1949. Mineral nutrition in relation to the biochemistry and physiology of potatoes. I. Effect of nitrogen, phosphate, potassium, magnesium and copper nutrition on the tyrosine content and tyrosinase activity with particular reference to blackening of tubers. Plant and Soil 2: 59-121.
11. van der WAAL, G. A. 1929. The "blueing" of potatoes (Trans. title). Tijdschr. over Plantenziekten 35: 60-68. (Abst.) R.A.M. 8: 459.
12. WIANT, J. S. 1945. Internal black spot of Long Island potato tubers. Amer. Potato Jour. 22: 6-11.

SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE

RESPONSE OF POTATOES TO MONOZOOSPORE LINES OF PHYTOPHTHORA INFESTANS[1]

Jack R. Wallin[2]

Summary

A technique for isolating monozoospores of Phytophthora infestans is described.

Differences in pathogenicity were noted among 9 monozoospore lines from potato Culture 1 cross-inoculated on 9 potato varieties. Moreover, the lines differed in their capacity for sporulating on certain potato varieties.

All lines from potato Culture 1 produced necrotic spots on two resistant potato varieties, Kennebec and Cherokee. However, none of the lines sporulated on these two varieties. Line 1-18 was the most virulent on Kennebec.

The 10 monozoospore lines from Culture 58 Line 12 were pathogenically alike on 8 potato varieties. All lines attacked and sporulated on 7 of the 8 varieties. A variety, 3XX-1, that was free from necrotic spots has the genotype $R_1 R_2 R_4$.

The 10 monozoospore lines from Line 12 were more virulent than those from Culture 1 in that they sporulated on Kennebec, Cherokee, and I803-3.

During the past 9 years, 111 isolates of Phytophthora infestans have been obtained from tomatoes and/or potatoes grown in Iowa, Illinois, Indiana, Nebraska, Minnesota, Michigan, North Dakota and Wisconsin. Some of these isolates were studied in cross-inoculation trials and were found to vary in their pathogenicity on susceptible potato and tomato plants (3). These findings suggested studies of the pathogenicity of monozoospores from within individual potato and tomato isolates. Hence, monozoospore lines[3] were obtained from each of two common parent isolates and cross-inoculated on Cobbler potato and Rutgers tomato. Under certain temperature variations these lines differed in their pathogenicity (4). In 1955 (1), pathogenic variation on selected potato genotypes was noted among 100 monozoospore lines obtained from a parent isolate identified as Race $1^t.4$. Ninety-six of the 100 produced lesions and sporulated on the hosts used while the remaining four produced flecks only. This difference in pathogenicity was attributed to a loss in virulence resulting from frequent transfer on culture media and not to variation among the lines.

These findings suggested a study of the pathogenicity of monozoospore lines from at least two common parent cultures on the potato late blight differential varieties[4]. Hence, 9 varieties were inoculated with each of 9 monozoospore lines isolated from parent Culture 1, and 8 varieties with each of 10 monozoospore lines isolated from a culture of monozoospore Line 12 from Culture 58. The results obtained are presented herein.

---

[1]Journal Paper No. J-3164 of the Iowa Agricultural Experiment Station, Ames, Iowa, Project 1163. Report of an investigation conducted under a regional project of the Research and Marketing Act (RMA Title II) in cooperation with the Iowa Agricultural Experiment Station, the Crops Research Division, United States Department of Agriculture, the North Dakota State Seed Department, and the North Dakota Agricultural Experiment Station. Dr. William G. Hoyman at North Dakota State College conducted all of the cross inoculation trials in his "late blight chamber". He generously donated his time and equipment to obtain the results presented herein. Dale N. Polhemus, Biological Aid, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, assisted in the laboratory work.

[2]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[3]Monozoospore line will be used throughout to designate the population resulting from a monozoospore.

[4]Variety will include unnamed potato selections that are designated by numbers.

MATERIALS AND METHODS

Nine monozoospore lines were obtained from Culture 1 isolated from a North Dakota Triumph tuber in 1947.

In July 1950, a potato plant in a potato-tomato plot at Lafayette, Indiana was inoculated with Culture 1. In August, the pathogen was recovered from a tomato fruit in the plot and was designated Culture 58. In greenhouse cross-inoculation trials on susceptible potatoes and tomatoes, monozoospore Line 12 was the most virulent of the 20. Therefore, 10 monozoospore lines were obtained from Line 12 for the present study.

The monozoospore lines from Culture 1 were cross-inoculated on potato varieties ND 3295-6, ND 3296-3, I803-3, ND 3297-2, B3131-8, B2876-1, Red Pontiac, Cherokee, and Kennebec.

The monozoospore lines from Culture 58 Line 12 were cross-inoculated on B595-76, B3131N2, 3XX-1, I803-3, Kennebec, Cherokee, Red Pontiac, and Early Gem. In previous experiments, these varieties varied in their reaction to Phytophthora infestans.

The monozoospores were isolated as follows: Sporangia from a 14-day-old culture growing on autoclaved peas were transferred to a flask of sterile, glass-distilled water and placed at 59° F until swimming zoospores were abundant. Thereupon, the zoospores were streaked with a nichrome wire loop upon plain agar in Petri dishes. The Petri dishes were placed overnight at 59° and examined the next morning under the microscope. Germinated zoospores, well removed from their neighbors, were marked for transfer. Next, the bottom portion of the dish was inverted on a microscope stage that had been wiped with alcohol. A marked zoospore was centered, removed from beneath the stage with a sterilized tungsten wire loop 380 microns in diameter and placed on soybean meal-dextrose agar in a Petri dish. The zoospore was incubated at 59° for 2 weeks. Next, the isolate was transferred to autoclaved peas in a 150-ml flask and incubated at 59°.

Inoculum was prepared by flooding 14-day-old pea cultures with glass-distilled water, decanting the liquid and filtering it through cheesecloth and incubating the sporangial suspension at 53° F for 2 1/2 hours. After this period swimming zoospores were abundant.

Potato leaves, each having five or more leaflets, were removed from the middle portions of plants growing in 8-inch pots in the greenhouse. The petiole of each detached leaf was placed in a test tube of tap water and slanted on damp sphagnum moss held in a greenhouse flat. Each of the varieties was placed in the same flat and inoculated at the same time. The inoculum was sprayed on the upper surface of the leaflets of each variety until the entire surface was wet. A separate atomizer was used for each culture. The flat was placed in a controlled temperature-relative humidity chamber (2) in which the temperatures ranged between 68° and 72° F and the relative humidity was 100 percent.

Varietal reaction was observed daily, and the final readings of lines from Cultures 1 and 12 were taken after 11 and 10 days respectively.

Blight reaction was evaluated as follows: 0 = no necrotic spots, 1 = very few necrotic spots, 2 = few necrotic spots, 3 = many necrotic spots, 4 = general necrosis. The following key was used to denote the quantity of spores on the lesions: 0 = none, 1 = slight and 2 = moderate.

In the trial involving the monozoospore lines from Culture 58, Line 12, the reaction was scored as plus or minus in the absence of visible differences in pathogenicity among the lines. Either they caused necrosis and sporulated or they produced no necrosis, spots or flecks. There were no intermediate stages to score.

RESULTS AND CONCLUSIONS

Monozoospore Lines from Culture 1.

The results (Table 1) obtained with 9 monozoospore lines from parent Culture 1 indicated some pathogenic variation among lines. For example, 1-35 and 1-37 were more pathogenic on ND3297-2 and B2376-1 than the other lines. Earlier tests showed that B2376-1 was highly resistant to race 0 isolates and the data in Table 1 indicated that this variety was more resistant than Cherokee or Kennebec. Culture 1 has been identified as race 1.

Only 1-34 failed to sporulate on ND3297-2, while Lines 1-35 and 1-37 sporulated more rapidly on this variety. All of the lines produced necrotic spots on Cherokee and Kennebec, the two varieties considered highly resistant to Phytophthora infestans. Lines 1-18, -23, -31, -35, and -37 were the most virulent on Cherokee, while 1-18 was the most virulent on

Table 1. Evaluation of pathogenicity of 9 monozoospore lines from Culture 1 on detached leaves of 9 potato varieties.

Variety	Monozoospore Line																	
	1-18		1-19		1-23		1-31		1-32		1-33		1-34		1-35		1-37	
	R.[a]	S.[b]	R.	S.	R.	S.	R.	S.	R.	S.	R.	S.	R.	S.	R.	S.	R.	S.
ND3295-6	4[c]	1	4	1	4	1	4	1	4	1	4	1	4	1	4	1	4	1
I803-3	3	0	2	0	3	0	3	0	3	0	3	0	3	0	3	0	3	0
ND3297-2	3	1	3	1	3	1	3	1	3	1	3	1	3	0	4	1	4	1
Red Pontiac	4	2	4	2	4	2	4	2	4	2	4	2	4	2	4	2	4	2
ND3296-3	1	0	1	0	3	0	1	0	2	0	2	0	1	0	2	0	3	0
Cherokee	3	0	2	0	3	0	3	0	2	0	2	0	2	0	3	0	3	0
B3131-8	3	0	1	0	3	0	3	0	3	0	3	0	3	0	2	0	3	0
Kennebec	3	0	1	0	1	0	1	0	1	0	1	0	2	0	2	0	2	0
B2876-1	1	0	1	0	0	0	1	0	1	0	1	0	1	0	2	0	2	0

[a]Blight evaluation
0 no necrotic spots
1 very few necrotic spots
2 few necrotic spots
3 many necrotic spots
4 general necrosis

[b]Sporulation values
0 no spores
1 few spores
2 moderate spores

[c]The mean reaction on 5 leaflets.

Table 2. The pathogenic response of 10 monozoospore lines obtained from monozoospore Line 12 from parent Culture 58 on 8 potato varieties.

Variety	Monozoospore line									
	12-1	12-2	12-3	12-4	12-5	12-6	12-7	12-8	12-9	12-10
Kennebec	+[a]	+	+	+	+	+	+	+	+	+
Cherokee	+	+	+	+	+	+	+	+	+	+
Red Pontiac	+	+	+	+	+	+	+	+	+	+
I803-3	+	+	+	+	+	+	+	+	+	+
Early Gem	+	+	+	+	+	+	+	+	+	+
3XX-1	-[b]	-	-	-	-	-	-	-	-	-
B595-76	+	+	+	+	+	+	+	+	+	+
B3131N2	+	+	+	+	+	+	+	+	+	+

[a] + = necrosis and sporulation on 5 or more leaflets.
[b] - = no necrotic spots on 5 or more leaflets.

Kennebec.

Line 1-23 differed from the other lines in that it did not produce necrotic spots on B2876-1.

These results suggested that monozoospore lines from the same parent isolate had different pathogenic capabilities. Possibly, the field isolate is a composite population comprised of individuals with different pathogenic capabilities.

Monozoospore Lines from Culture 58, Line 12

The monozoospore lines obtained from Culture 58 Line 12 were applied to the detached leaves of eight potato varieties in the same manner described previously.

As shown in Table 2, the pathogenic response of the lines was similar on the eight varieties.

All of these varieties except Red Pontiac and Early Gem have been considered resistant to race 0 of the late blight fungus. The absence of necrotic spots on 3XX-1 was attributed to the presence of $R_1 R_2 R_4$ genes.

Lines 12-5 and 12-10 differed from the others in their behavior on B3131N2. These two lines sporulated on the tenth day after inoculation, 5 days later than the other lines.

These results indicated that monozoospore lines from within a monozoospore line were pathogenically homogeneous. The only indication of variation in behavior among these lines was the difference in time required for two of them to sporulate on one potato variety.

Another fact worthy of note was the sporulation of all the monozoospore lines from Line 12 on Kennebec, Cherokee, and I803-3. This fact suggests that potato race 1 was altered by passage through tomato in the field so that either another race was obtained or a more virulent population of the same race developed. Incidentally, the monozoospore lines from Culture 1 did not sporulate on these varieties. Apparently, the lines from monozoospore Line 12 were considerably more pathogenic than those from Culture 1.

Literature Cited

1. GRAHAM, K. M. Distribution of physiological races of Phytophthora infestans (Mont.) de Bary in Canada. 1955. Amer. Potato Jour. 32: 277-282. 1955.

2. HOYMAN, Wm. G. 1956. A large inoculation chamber with automatic temperature and humidity controls. Amer. Potato Jour. 33: 214-217.

3. WAGGONER, PAUL E., and J. R. WALLIN. 1952. Variation in pathogenicity among isolates of Phytophthora infestans on tomato and potato. Phytopathology 42: 645-648.

4. WALLIN, JACK R. Annual report of the crop disease forecasting project in the North Central States, 1954-1955. Unpublished.

IOWA AGRICULTURAL EXPERIMENT STATION, UNITED STATES DEPARTMENT OF AGRICULTURE, AGRICULTURAL RESEARCH SERVICE, NORTH DAKOTA STATE SEED DEPARTMENT, AND NORTH DAKOTA AGRICULTURAL EXPERIMENT STATION

THE RELATION OF RAINFALL AND TEMPERATURE TO LATE BLIGHT OF POTATO IN NORTHERN OHIO

R. A. Hyre and J. D. Wilson

Abstract

The relation of rainfall and temperature to late blight of potato (due to Phytophthora infestans (Mont.) D By) in northern Ohio was determined by means of "moving" graphs. Hypothetical forecasts by means of 5-day temperature and 10-day rainfall graphs would seem to be somewhat less accurate than those reported, using a similar method, for the upper Atlantic Coastal States.

INTRODUCTION

In 1948 cooperative programs were undertaken between various States and the United States Department of Agriculture to establish facilities for forecasting certain plant diseases in regional areas of the United States. To forecast late blight of potato (caused by Phytophthora infestans (Mont.) D By) in northeastern United States Hyre and co-workers (2, 3, 4) found a "moving graph" method, calculated from rainfall and temperature data supplied by the United States Weather Bureau, to be preferable to a method using relative humidity and temperature data which were obtained from hygrothermograph charts. In Iowa and surrounding States, Wallin and co-workers (6, 7, 8, 9) found a "cumulated" rainfall method to be unsatisfactory. The "cumulated" rainfall method, likewise, was found to be unsatisfactory in the Northeastern States (2).

In the present paper rainfall, temperature, and blight data from northern Ohio were analyzed to see whether or not the "moving" graph method might be satisfactory for predicting late blight in that area.

METHODS

Precipitation and temperature data for Akron and Wooster, Ohio, were obtained from Climatological Data for the years 1905 through 1956. An average of the data for the two cities was used except in the limited number of cases when data were missing for either of these two cities and those of Canton were substituted.

Records of the occurrence of late blight of potato in the Akron-Wooster area were obtained from several sources. Tilford (5) lists the amount of blight in Ohio from 1905 through 1942. His source of information was the files of the Ohio Agricultural Experiment Station at Wooster. In addition to the records at Wooster blight records were obtained from Ohio State University (through the courtesy of Dr. Blair Janson) and the United States Department of Agriculture Plant Disease Reporter. However, some years were not included in the analysis, because either of the uncertainty of blight in the Akron-Wooster area even though it was reported in Ohio, or of a conflict in records. The remaining years were grouped according to whether blight was rated severe, moderate, light or none.

The starting date of the forecasting season was determined by plotting average precipitation and temperature for 16 non-blight and 4 severe-blight years, according to the method of Cook (1). The days "favorable" for blight during each season were then calculated. A day was considered favorable for blight if both the temperature and rainfall were favorable. The temperature was considered favorable if the mean for 5 days, ending that day, was less than 78° F. However, any day was considered unfavorable if the minimum temperature for that day was less than 45° F. Rainfall was considered favorable if the 10-day total, ending that day, was 1.2 inches or more. The initial occurrence of blight would be forecast after ten consecutive blight-favorable days and a weather forecast at that time of continuing blight-favorable weather. The disease would be expected about 2 weeks after it was forecast.

RESULTS

To determine the start of the forecasting season, average 7-day temperature and total 7-day precipitation for 16 non-blight years (1909, 1910, 1911, 1916, 1918, 1919, 1921, 1922, 1923, 1927, 1930, 1936, 1944, 1952, 1953 and 1954) and for 4 severe blight years (1905, 1915,

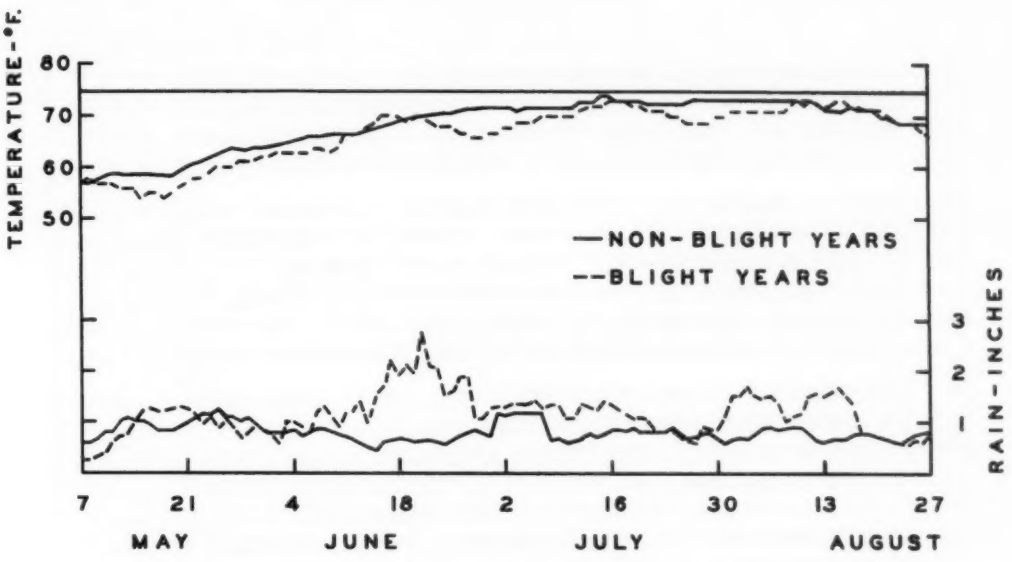


FIGURE 1. Comparison of the rainfall and temperature for Akron and Wooster, Ohio, for 16 non-blight and 4 severe-blight years. Each point on the rainfall lines represents the total rainfall for the 7-day period ending that day, and each point on the temperature lines represents the average of the mean temperature of the days during the 7-day period.

1920, and 1938) were plotted (Figure 1). The rainfall lines diverged on June 7 with the greater rainfall for the severe blight years. Since these were 7-day figures the blight forecasting period would start June 1. The temperature lines had no part in determining this date since both lines remained below 75° F throughout.

The days favorable for blight during the various growing seasons were calculated and the years were grouped according to the severity of blight (Figure 2). In general the seasons during which blight was most severe also had most 10-day periods favorable for it, and blight was light or absent during seasons with few periods favorable for it. However, there were exceptions to this general rule. In 1909, for example, there were several blight-favorable periods but no blight was recorded.

The causal relationships between favorable periods and the amount of blight were arbitrarily checked as being correct or incorrect depending on the presence (and number) or absence of disease-favorable periods and the amount of blight recorded. All "severe" and "moderate" blight years were rated "correct". Rated "incorrect" were years 1935, 1941, and 1943 for the "light" blight years, and 1909, 1916, 1919, 1927, 1952, and 1953 for the non-blight years. This would give an overall correct rating of 78 percent. It should be noted that the ratings were all "correct" for the years when blight was recorded as being severe or moderate in amount.

For comparison other criteria were used for determining blight-favorable periods. They were 7-day mean temperature of 77° F and 10-day rainfall of 1.0, 1.1, or 1.2 inches; and 7-day mean temperature of 78° F and 10-day rainfall of 1.2 inches. In all cases the overall rating of the relationship of blight-favorable periods to the amount of blight was less accurate than the 78 percent when using criteria of 5-day mean temperature of 78° F, with any day unfavorable if the minimum temperature was less than 45° F, and 10-day rainfall of 1.2 inches. The ratings were 53 to 70 percent correct with other than the adopted standards for determining blight-favorable periods.

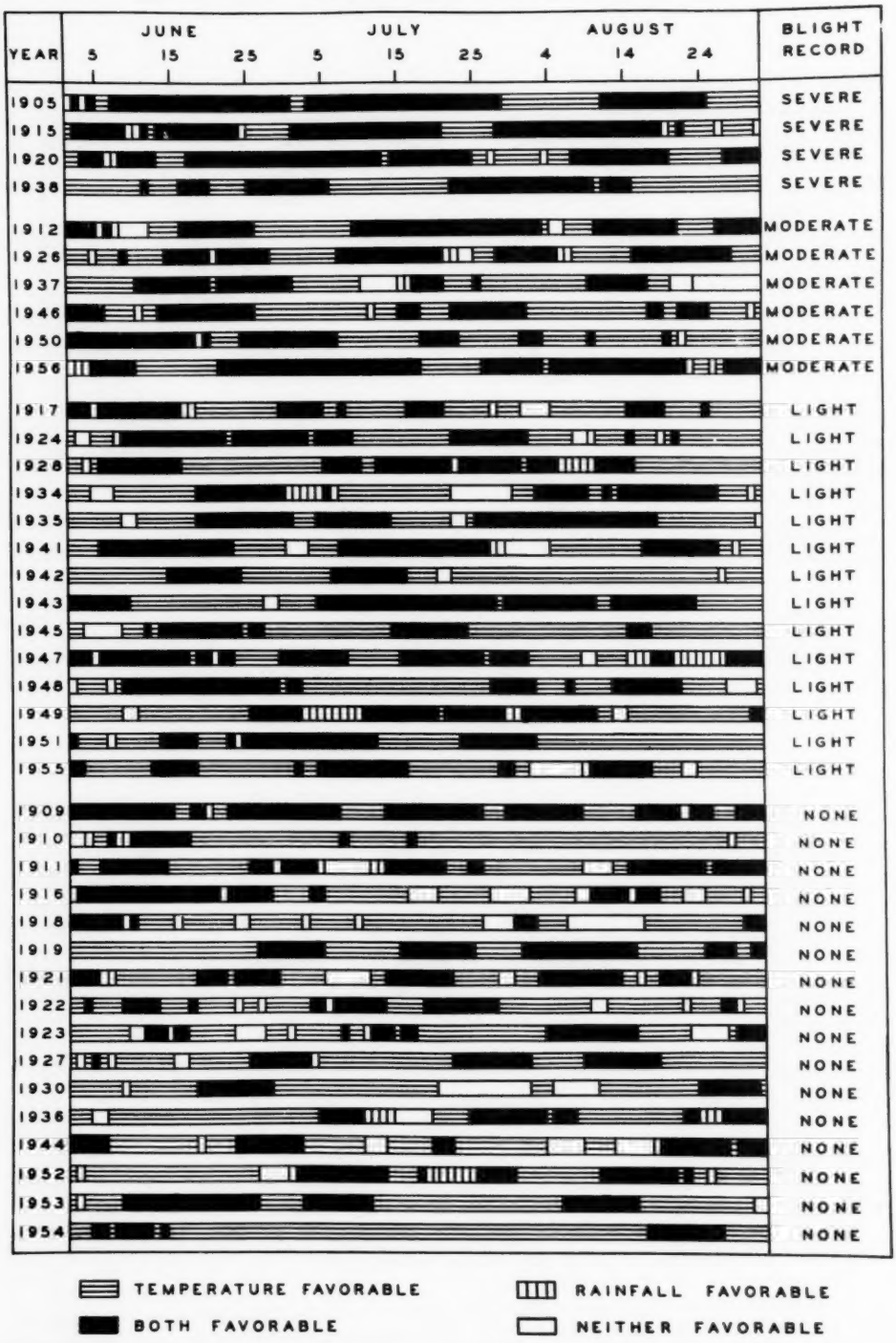


FIGURE 2. Summary of the rainfall and temperature data for Akron and Wooster, Ohio, in relation to the importance of late blight.

DISCUSSION AND SUMMARY

The relationship of blight-favorable periods with late blight of potato in the Wooster area was good for the years of severe or moderate amounts of blight. It was not so good for the years of little or no blight. In the latter years forecasts of blight, if they had been made, would have been for blight when it did not occur and not for "no blight" when it would have occurred. Hypothetical forecasts, by the method described here, would seem to have been less accurate for the Wooster area than was reported for the upper Atlantic Coastal States.

Literature Cited

1. COOK, HAROLD T. 1949. Forecasting late blight epiphytotics of potatoes and tomatoes. Jour. Agr. Res. 78: 545-563.
2. HYRE, R. A. 1954. Progress in forecasting late blight of potato and tomato. Plant Dis. Reptr. 38: 245-253.
3. HYRE, R. A. 1955. Three methods of forecasting late blight of potato and tomato in northeastern United States. Amer. Potato Jour. 32: 362-371.
4. HYRE, R. A., and REINER BONDE. 1955. Forecasting late blight of potato in northern Maine. Amer. Potato Jour. 32: 119-125.
5. TILFORD, PAUL E. 1943. Factors favoring spread and control of late blight. Proc. Ohio Veg. and Potato Grow. Assoc. 28: 7-12.
6. WALLIN, J. R., and Wm. G. HOYMAN. 1954. Forecasting potato late blight in North Dakota. North Dakota Agr. Exp. Sta. Bimonth. Bul. 16: 226-231.
7. WALLIN, J. R., and R. W. SAMSON. 1953. The relation of cumulative amount and frequency of rainfall and mean temperature to late blight in Indiana. Amer. Potato Jour. 30: 262-270.
8. WALLIN, J. R., E. K. WADE, and H. M. DARLING. 1953. The relation of weekly mean temperatures and cumulative rainfall to late blight epiphytotics in Wisconsin. Amer. Potato Jour. 30: 231-242.
9. WALLIN, J. R., and PAUL E. WAGGONER. 1949. The influence of weekly cumulative rainfall and temperature on potato late blight epiphytotics in Iowa. Plant Dis. Reptr. 33: 210-218.

CROPS RESEARCH DIVISION, UNITED STATES DEPARTMENT OF AGRICULTURE; AND THE OHIO AGRICULTURAL EXPERIMENT STATION, WOOSTER, OHIO

REACTIONS OF WATERMELON PLANT INTRODUCTIONS AND VARIETIES TO DOWNY MILDEW IN NORTH CAROLINA

N. N. Winstead, Robert Aycock, and J. M. Jenkins, Jr.[1]

Summary

Approximately 300 watermelon plant introductions were evaluated for resistance to downy mildew in 1954. Introductions that were resistant and certain other varieties and breeding lines were tested again in 1956. Plant Introductions 179660 and 179875 were highly resistant and Plant Introductions 169289, 169290 and 171392 were somewhat less resistant in both tests. Plant Introductions 189225, 164460 and 183399 were rated as moderately resistant, resistant, and resistant, respectively, in 1954 but were not tested in 1956. The plants of the Royal Golden variety were also resistant or tolerant to this disease. All other varieties and lines and plant introductions were susceptible.

Downy mildew (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.) frequently causes severe losses to watermelon in North Carolina. Since fungicides are difficult to apply and results are often unsatisfactory[2], resistance to downy mildew would be of great value to North Carolina watermelon growers.

Parris reported in 1951[3] that 12 strains of watermelon obtained from the Dominican Republic were highly resistant to P. cubensis. Although it was not possible to obtain seed of these lines, approximately 300 other plant introductions[4] and varieties were evaluated for susceptibility or resistance to downy mildew in this study.

Five hills of each variety were planted in the field in the summer of 1954 at the Horticultural Crops Research Station, Castle Hayne, North Carolina. In that year the downy mildew disease was severe. Plants were rated for resistance on the following basis: 0 = immune; 1 = resistant, few lesions and sparse sporulation; 2 = moderately resistant; 3 = susceptible, lesions numerous and sporulation abundant; 4 = severe.

None of the watermelons tested were immune; however, several lines were resistant. Plant introductions 179660 and 179875, rated as highly resistant, had very few lesions in which only a few spores were produced. Two others, 164460 and 183399, also were placed in the highly resistant class; however, a few more lesions were observed on leaves of these plants. Four introductions and 1 variety, 169289, 169290, 171392, 189225 and Royal Golden, were considered moderately resistant. All other lines were considered susceptible. Disease ratings of these varieties and plant introductions are given in Table 1.

Introductions found to be resistant in 1954 as well as additional varieties were tested in 1956. Plant Introductions 179660 and 179875 were highly resistant in 1956 as well as in 1954. Plant Introduction 171392 was also placed in the most resistant class. Plant Introductions 169289 and 169290 were again rated as moderately resistant. Other varieties and breeding lines tested in 1956 were resistant. The reactions of these lines in 1956 are given in Table 2.

---

[1] Assistant Professor of Plant Pathology, North Carolina State College, Raleigh, North Carolina; Associate Professor of Plant Pathology; and Research Professor of Horticulture, Horticultural Crops Research Station, Castle Hayne, North Carolina, respectively.

[2] Ellis, D. E. 1953. Cucurbit diseases in North Carolina and their control. North Carolina Agricultural Exp. Sta. Bul. 380. 11 p.

[3] Parris, G. K. 1951. Downy mildew resistant strains of watermelon from the Dominican Republic, West Indies. Plant Dis. Reptr. 35: 399-401.

[4] Obtained from Dr. Edwin James, Division of Plant Exploration and Introduction, Regional Plant Introduction Station, Experiment, Georgia.

Table 1. Reactions of watermelon varieties and introductions to Pseudoperonospora cubensis.

Entry	Disease index	Entry	Disease index	Entry	Disease index	Entry	Disease index	Entry	Disease index	Entry	Disease index
Black Diamond			4			Dixie Queen W R			4		
Calhoun Sweet			4			Fairfax			4		
Charleston Gray			4			Florida F 53			4		
Chris-Cross			4			Northern Sweet			4		
Congo			3			Purdue Hawksbury			4		
Dixie Queen Hybrid			4			Royal Golden			2		
161373	4	169234	4	169284	4	174099	4	176921	4	180426	4
162667	4	169235	4	169285	4	174100	4	176922	4	180427	4
163202	4	169236	4	169286	4	174101	4	177318	3	181740	4
163203	4	169237	4	169287	4	174104	3	177321	4	181741	4
163204	4	169238	4	169289	2	174105	4	177322	4	181742	4
163205	4	169239	4	169290	2	174106	4	177323	4	181743	4
163572	4	169241	4	169291	4	174107	4	177324	4	181744	4
163574	4	169242	4	169292	4	174108	4	177325	4	181935	4
164146	4	169243	4	169293	3	174812	3	177326	4	181936	4
164247	4	169244	4	169294	4	175102	4	177328	4	181937	4
164248	3	169245	4	169295	4	175651	3	177329	4	181938	4
164460	1	169246	4	169296	4	175652	4	177330	4	182175	4
164474	4	169247	4	169297	4	175653	4	177331	4	182176	4
164475	4	169248	4	169299	3	175655	4	178870	3	182177	3
164543	4	169249	4	169300	4	175657	4	178872	4	182178	4
164550	4	169250	4	171392	2	175659	3	178873	4	182179	4
164570	4	169251	4	171579	3	175660	4	178874	4	182180	4
164633	4	169252	4	171580	4	175661	4	178875	4	182183	4
164634	4	169253	3	171581	4	175662	4	178876	3	182932	4
164636	3	169254	4	171582	4	175663	4	179232	4	182933	4
164639	3	169255	4	171583	4	175664	4	179233	4	182934	4
164655	4	169256	4	171584	4	175665	4	179234	4	182935	3
164665	4	169257	4	171585	4	176485	3	179235	4	183022	4
164685	4	169258	4	171586	4	176486	4	179236	4	183023	4
164687	4	169259	4	171587	4	176487	4	179237	4	183033	3
164708	4	169260	4	172786	4	176488	4	179238	4	183123	4
164709	4	169261	4	172787	4	176489	4	179239	4	183126	4
164737	4	169262	4	172788	4	176490	4	179241	4	183217	4
164748	4	169263	3	172789	4	176491	4	179243	3	183299	4
164804	4	169264	4	172790	4	176492	4	179249	4	183398	4
164977	4	169265	3	172791	4	176493	4	179660	1	183399	1
164992	4	169266	4	172792	3	176494	4	179661	4	183673	3
164998	4	169267	4	172794	4	176496	4	179662	4	184800	3
165002	4	169268	4	172795	4	176497	3	179875	1	185030	3
165024	4	169269	4	172796	4	176499	4	179876	4	185635	4
165448	4	169270	4	172797	4	176905	4	179877	4	185636	4
165451	4	169271	4	172798	4	176906	4	179878	4	186974	4
165523	4	169272	4	172800	4	176908	4	179879	4	188808	4
166993	4	169273	4	172801	3	176909	4	179880	4	189225	2
167026	4	169274	4	172802	4	176910	4	179881	3	189318	4
167045	4	169275	4	172803	4	176911	4	179882	4	190050	4
167059	4	169276	4	172804	4	176912	4	179883	4	192937	4
167124	3	169277	4	172805	4	176913	4	179884	4	192938	4
167125	3	169278	4	173667	4	176914	4	179885	4	193490	4
167126	3	169279	4	173668	4	176915	3	179886	4	193963	4
167219	4	169280	4	173669	4	176916	4	180275	4	193964	4
167222	4	169281	4	173670	4	176917	4	180276	4	193965	4
169232	3	169282	4	173888	4	176918	3	180277	4	195562	4
169233	4	169283	4	174098	4	176919	4	180278	4	195928	3
										197416	4

Table 2. Reactions of watermelon varieties and introductions to Pseudoperonospora cubensis in 1956.

Entry	Disease Index	Entry	Disease Index
Dominican Republic	4	Takii Gem	4
Black Diamond	4	Congo	4
Chris-Cross	4	W-453	3
White Hope	4	51-23W	3
Miss. 1717	4	169289	2
Ark. 55-11	4	169290	2
Georgia 4	4	171392	1
La. 1	4	179660	1
NC 1956-126	3	179875	1

NORTH CAROLINA STATE COLLEGE, RALEIGH, AND HORTICULTURAL CROPS RESEARCH STATION, CASTLE HAYNE, NORTH CAROLINA

PHACIDIOPYCNIS (PHOMOPSIS) CANKER AND DIEBACK OF CONIFERS

Glenn Gardner Hahn[1]

Abstract

The study concerns a canker-dieback disease in plantations of exotic conifers, particularly Douglas-fir and larch. The causative agent, a wound parasite, was described originally on saplings or poletimber of Douglas-fir in 1920 in Scotland. Identified as so-called Phomopsis pseudotsugae, it should now be known as Phacidiopycnis pseudotsugae, the imperfect stage of a new species Phacidiella coniferarum. The pathogenicity of P. pseudotsugae was first proven by inoculation experiments conducted by the writer in Scotland; his results were confirmed subsequently by independent investigations by pathologists in Holland. Symptoms of the disease on different conifers under conditions of natural infection are described. These refer particularly to the disease on exotic Douglas-fir in Europe. In the United States the disease occurs only in the Northeast where it is now identified with the "Phoma disease" of eastern white-pine saplings. It is not known to occur in native Douglas-fir stands. The complete life cycle of P. pseudotsugae has been found to date only on Pinus strobus in the United States. Although its American origin is suspect, this would appear to be highly uncertain because of the wide geographical and host range of the imperfect stage. These data are important for Federal Quarantine purposes. Positive inoculation results under Connecticut conditions with the pathogen are described on young plantation stock of Abies fraseri, Larix leptolepis, Picea abies, P. glauca, P. pungens, Pinus strobus, Pseudotsuga menziesii, P. menziesii, var. glauca, and Tsuga canadensis. Infection occurred during the autumn and early spring but not during the summer. Reference is made to the author's foreign inoculation studies of P. pseudotsugae in which infection tests of young Douglas-fir did succeed under summer conditions in Scotland. Damage due to the parasite on plantation trees and natural reproduction of native white pine is discussed. Procedures to be followed in effecting control in the Northeast are outlined.

INTRODUCTION

Since its discovery by Wilson on sapling Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) in Scotland, the so-called Phomopsis disease of conifers has received much attention from foresters abroad (18). It still remains primarily a plantation disease of exotic conifers, the investigation of which continues to attract the attention of forest pathologists in western Europe (16, 22, 23).

The causal organism was originally described as Phomopsis pseudotsugae by Wilson (17) and has been known subsequently by that name. It is now known to be the imperfect stage of the ascomycete, Phacidiella coniferarum Hahn, which the writer discovered on cankered white-pine saplings (Pinus strobus L.) in Maine (9). The pycnidial stage of Phacidiella is Phacidiopycnis; hence the new combination, Phacidiopycnis pseudotsugae (M. Wils.) Hahn (9). As this is the stage encountered on cankers in foreign countries as well as most frequently in this country, the disease accordingly is linked with it rather than with the rarely occurring Phacidiella stage. The distribution and present known hosts of P. pseudotsugae throughout the world--important for Federal quarantine purposes -- are now known (9).

After the first announcement in Scotland of the canker and dieback of typical Douglas-fir, proof was needed to demonstrate that the attributed parasite, Phomopsis pseudotsugae, is the actual causative agent. Its taxonomy and relationship to allied fungi (with which it was confused) on Douglas-fir and other conifers also needed clarification. In addition, information was needed on the factors that affect the spread, intensification, and control of the disease under foreign

[1] Pathologist, Forest Insect and Disease Laboratory, Northeastern Forest Experiment Station, Forest Service, United States Department of Agriculture, New Haven, Connecticut. Harold G. Eno, also of the Forest Insect and Disease Laboratory at New Haven, assisted in the pathological investigations made in this country, and contributed one of the illustrations.

conditions. Such information would provide a basis for control operations if the then potentially menacing disease were introduced eventually into this country (2, 13). Accordingly, in 1926 to 1929 the writer was assigned by the United States Department of Agriculture to conduct investigations in Great Britain in cooperation with Dr. Malcolm Wilson, formerly mycologist, University of Edinburgh and the British Forestry Commission.

Results of the writer's taxonomic studies regarding the identity of the Douglas-fir canker parasite have been published (7, 8, 9). Moreover the parasitism of Wilson's Phomopsis pseudotsugae was proved under controlled experimental conditions. A brief preliminary notice of positive inoculation results was reported by Wilson and Hahn (21). This announcement was supplemented by Wilson (20) with a discussion of the Scottish inoculation results in relation to frost injury. Independent European investigations carried on subsequently in Holland by Koning (10) and Van Vloten (16) have corroborated the writer's results in Great Britain, confirming the pathogenicity of the Douglas-fir pest.

The resurgence of forest-tree planting in Great Britain and continental Europe after World War I has been accompanied by diseases on North American trees planted in foreign countries (15). One of these was the Phacidiopycnis disease that occurs on the extensively planted Douglas-fir from the Pacific Coast. Because this host proved to be most susceptible, the symptoms described here refer particularly to the disease on this exotic in Europe. Results of the pathological investigations of the parasite under American conditions are presented for the first time.

ORIGIN OF THE PARASITE

Inasmuch as the complete life cycle of Phacidiopycnis pseudotsugae (syn. Phomopsis strobi Syd.) is known only on eastern white pine in the Northeast (9), the question presents itself as to whether the parasite is native to this country and has been introduced elsewhere. Birch (1) in New Zealand was inclined to believe that it had been introduced into Australasia on tree seed of American origin. This opinion was strengthened by the fact that prior to the period when P. pseudotsugae assumed economic importance as a pathogen on exotic conifers there apparently was no record of it in the Dominion. On the other hand, E. Rostrup collected the organism under another name as far back as 1884 on the exotic Pinus strobus in Denmark as well as on mountain pine, P. mugo Turr. (8).

In view of our present knowledge of the wide geographical and host range of P. pseudotsugae, its origin would appear to be highly moot. Despite the fact that introduced Douglas-fir is the host that first gave it pathological prominence, P. pseudotsugae is still unknown in native Douglas-fir stands in this country (9).

NATURAL INFECTION

Although canker and dieback caused by Phacidiopycnis pseudotsugae occur on older trees, the disease is essentially one that affects saplings or poletimber. Infection takes place through wounds during the period from dormancy to active growth in the spring. The intensity of the disease is high when infection occurs early in the resting period of the trees. However, its attack becomes limited by the activity of the trees during the growing season. The formation of periderm as a result of cambial activity in connection with fungus infection provides a complete barrier to localize the spread of the hyphae of the parasite.

On Douglas-Fir

In European plantations, Douglas-fir of West Coast provenance has shown itself to be generally more susceptible to the Phacidiopycnis disease than the blue or Colorado variety of the species (Pseudotsugae menziesii var. glauca (Biessn.) Franco). In Sweden, however, Lagerberg (11) stated that the disease occurred mainly on the blue variety.

Three types of infection that cannot be sharply delimited are recognized on exotic Douglas-fir:

1. Non-girdling Bark Canker -- Cankers of this type form generally on trees older than 10 years, on the main trunk or on the larger branches (more than 2 cm in diameter). Cankers rarely exceed 20 cm in length, their size and abundance depending not only on the age of the infected host tissue but also on certain other factors that predispose the trees to disease, e.g., young trees growing on an unfavorable site. The extension of dead tissue above and

below the point of infection is generally equal and is proportionally greater than the lateral extension. Secondary cork eventually surrounds the infected area in a continuous layer, and this prevents further spread of the parasite. Abnormal wood is also produced all around the edge of the canker. Eventually a well-developed callus forces off the diseased and degenerate cortical tissues as a superimposed plate or shield. This type of injury, which Boyce (4) has suggested should be termed "bark anthracnose", results in arrested annual cankers.

2. Girdling Canker -- Terminal shoots or branches 1 to 2 cm in diameter and 3 to 6 years old are completely and rapidly girdled by this type of canker at the point of infection. The spread of the parasite at both the lower and upper limits of infection, as in the case of bark cankers, is checked by secondary cork barriers. The girdled area becomes discolored, desiccated, and sunken. The uninfected tissues above and below the girdle become swollen, the swelling above being generally more pronounced and larger. Ultimately the uninfected shoot above the constricted and diseased part dies. German pathologists refer to this disease type as "Einschnürungskrankheit"; the Dutch call it "Insnoeringsziekte."

3. Dieback -- This type of infection occurs generally toward the apex of slender terminals of either the main axis or lateral branches that have a diameter of approximately 0.5 to 1.5 cm. Growth 1 to 2 years old, but occasionally 3 to 4 years old, is attacked. The smaller diameters are girdled quickly at the point of infection, but Phacidiopycnis does not spread far below it. Although a layer of secondary cork forms irregularly across the cortex, delimiting the disease below the infection site, it does not form above. The girdled shoot, at times measuring 30 cm or more in length, becomes desiccated and dead. This infection type is not actually dieback, but rather a gradual dying of tissue above the infection locus. When infection occurs in close proximity to the apex itself, the resulting symptoms look more like typical dieback. In both cases the downward extension of the pathogen is checked. Whether the tissues die in a downward or upward direction, the final appearance is the same. For the reasons stated, it is proposed to retain the term dieback for this type of disease.

In both canker and dieback, erumpent sporulating fruiting bodies (pycnidia) of Phacidiopycnis pseudotsugae occur in abundance. In dieback, however, they are localized around the seat of infection and do not occur beyond in the dead dry tissues that result from girdling. Observations made in Scotland by the writer showed that the dessiccated tissues are colonized by saprophytic fungi, e.g., species of Sclerophoma or Cytospora, Phomopsis occulta Trav., or P. conorum (Sacc.) Died. (7, 8).

As previously stated, Phacidiopycnis pseudotsugae has not been found in native Douglas-fir forests. However, it has been found -- but not commonly -- in plantations of the blue variety of Douglas-fir in the northeastern United States. In these instances it was observed to cause girdling or non-girdling cankers of the smaller branches.

On Larch

The disease occurs on planted stock of both European (Larix decidua Mill.) and Japanese (L. leptolepis (Sieb. et Zucc.) Gord.) larch in Europe. On the latter host it is now receiving from foreign pathologists the attention that was formerly accorded it on Douglas-fir (4, 16, 23). Bark cankers are common on 10- to 20-year-old stock of this conifer. Their vertical extent is much greater than that of cankers on Douglas-fir. Moreover, they differ further in that the margins of the cankers on larch are marked by pronounced resinous exudations; the diseased cortical tissues crack, and as a consequence are not cast off as plates. The healing process that follows infection is not so rapid as in Douglas-fir and often is not completed (18). Cankers on branches of Japanese larch 45 to 50 feet above the ground have been reported in Scotland (20).

In the Northeast, the disease on introduced Japanese and European larch in old plantations in Massachusetts is confined to resinous girdling or non-girdling cankers on branches of small diameter. In addition, Phacidiopycnis pseudotsugae was observed fruiting as a saprophyte on uncankered dead suppressed larch branches. Van Vloten (16) reported the same condition in Japanese larch plantations in Holland. His experiments demonstrated that a great quantity of pycnidia and spores produced on dead branches left in a stand functioned as natural inoculum for the infection of wounds.

FIGURE 1.
A girdling canker, about 3/4 natural size, on the trunk of an eastern white pine sapling infected naturally with Phacidiopycnis pseudotsugae. Pycnidia of the parasite occur in the canker.

FIGURE 2.
A non-girdling basal canker, about 1/4 natural size, on a mechanically-injured living white pine sapling. Both the Phacidiella and Phacidiopycnis stages of the pathogen occur in the canker.

On Pine

In Europe there are no reports of Phacidiopycnis pseudotsugae cankering the trunks and branches of pines; the fungus has been found only on small shoots and needles (8). In New Zealand, however, cankering of sapling pine exotics was reported by Birch (1). He described the following infection types: shoot dieback originating from infected frost cracks; cankers on the main axis resulting from the infection of snow-weakened tissue at the juncture of trunk and lateral, or from infection of the trunk from an infected lateral; and occasional bark cankers on the trunks of older trees.

In the Northeast of the United States the disease on sapling eastern white pine has been present for many years. In the literature it was called the "Phoma disease", which was reported in connection with the early investigations of white pine blister rust (Cronartium ribicola A. Fisch.) (12). It appears as cankers of the girdling type occurring on the main axis of young reproduction (Fig. 1) or as cankers of non-girdling type on the trunks of older stock (Fig. 2). The disease is not to be confused with basal girdling cankers that are caused by ant injury on young white-pine reproduction (6). Infections of Phacidiopycnis pseudotsugae have also been observed on the smaller branches of mature pines 50 feet or more above the ground. Fruiting bodies of the parasite occur abundantly in the cankers as well as in the diseased bark of uncankered suppressed or windthrown trees. In the latter, P. pseudotsugae behaves saprophytically, growing in weakened tissues previous to their desiccation and final death.

On Other Host Genera

Natural infection of Phacidiopycnis pseudotsugae on species of Abies, Cedrus, Sequoia, and Tsuga in Europe (20) is only occasional; and nothing new with regard to infection types can be reported concerning them. However, Wilson (19, 20) reported a substantial dieback of shoots and branch cankers of the girdling type on the branches of middle-aged cedars in Great Britain, but bark cankers were not observed on the larger branches or on the trunk.

Although it will be shown in the following section that Canadian hemlock (Tsuga canadensis (L.) Carr.) is susceptible to the disease under experimental conditions, Phacidiopycnis pseudotsugae has not been collected on this host in the Northeast. However, occasional small branch cankers have been observed on balsam fir (Abies balsamea (L.) Mill.).

INOCULATIONS

The pathogenicity of Phacidiopycnis pseudotsugae under northeastern United States conditions was tested by inoculations of nursery trees in an established plantation at Cheshire, Connecticut. The hosts tested represented seven species of conifers, including Fraser fir (Abies fraseri Poir.), Norway spruce (Picea abies (L.) Karst.), white spruce (P. glauca (Moench.) Voss), Colorado blue spruce (P. pungens Engelm.), eastern white pine, Douglas-fir and its blue form, and Canadian hemlock. The trees were growing satisfactorily except for the white pine, which was heavily weeviled, and three poor specimens of Douglas-fir.

The inoculum consisted of mycelium and mature pycnidia of isolates of Phacidiopycnis in pure agar cultures. The isolates represented both American and European collections: those from the Northeast were obtained from cankered white pine and European and Japanese larch; those from Norway, from Norway spruce and Douglas-fir[2].

All inoculations were made by inserting small culture bits of the isolate to be tested into incisions on the trunk or branch; a tongue-like section of the cortex was cut with a sterile scalpel and the inoculum was placed under it. The inoculation site was then firmly secured by a wrapping of moist cotton over which there was a second wrapping of grade "M" parafilm. Growth 1 to 6 years old was tested. Control incisions on growth of similar age, placed either below the inoculation site or on separate branches, were identical with the inoculations except that sterile malt extract agar bits were inserted. All coverings were removed within a month after inoculation.

Three inoculation series were performed: the first was made during the summer of 1953; the second in early autumn of the same year when the trees were approaching dormancy; the third in the next year in the early spring before active growth had fully resumed. An inoculation was considered positive when a definite canker or dieback was produced. Fruiting of the parasite, which occurred frequently in the artificially produced lesions, gave further assurance of pathogenicity. In doubtful cases, diseased areas associated with the inoculation site were checked by re-isolating an organism identical with the original inoculum.

RESULTS

Summer Series -- A total of 840 inoculations, and 420 check incisions of the aforementioned hosts, were made during late June and early July. Fifteen isolates of Phacidiopycnis pseudotsugae were tested: five from white pine and European and Japanese larch in the United States (420 inoculations); five from Douglas-fir in Norway (228 inoculations); and five from Norway spruce in Norway (192 inoculations). Negative results were obtained: in all instances both inoculated and check incisions callused over and healed.

Autumn Series -- A total of 382 inoculations, together with 127 check incisions, were made on the test conifers used in the summer series. Isolates of Phacidiopycnis pseudotsugae previously tested were used in the October experiment; 222 inoculations were made with the American isolates and 160 inoculations with the Norwegian isolates. As shown in Table 1, infection was obtained on all the hosts with the isolates of both American and Norwegian origin.

---

[2] The writer expresses his appreciation to Dr. Hakon Robak, Forest Research Institute, Stend, Norway, not only for his continued interest and cooperation in the investigation but also for pure cultures of Phacidiopycnis pseudotsugae identified as the synonymous species, the blue stain fungus Discula pinicola (Naumov) Petr. from Norway spruce and Phomopsis pseudotsugae from Douglas-fir (Hahn, 1957). Robak has published the two fungus species as being identical (14).

Table 1. Results of autumn and spring inoculations on seven conifer species with isolates of Phacidiopycnis pseudotsugae*.

Species inoculated	Source of inoculum: United States (Northeast)						Source of inoculum: Norway				Total	
	Larix decidua		Larix leptolepis		Pinus strobus		Picea abies		Pseudotsuga menziesii			
	Autumn	Spring	Autumn	Spring	Autumn	Spring	Autumn	Spring	Autumn	Spring	Autumn	Spring
Abies fraseri	6/9	2/6	--	--	11/18	0/18	4/9	2/12	3/18	2/9	24/54	6/45
Picea abies	2/9	1/9	4/18	--	6/18	0/9	4/9	5/56	0/18	1/18	16/72	7/92
Picea glauca	--	--	--	--	2/9	0/9	4/15	5/15	--	--	6/24	5/24
Picea pungens	--	--	--	--	0/6	1/6	5/9	3/9	--	--	5/15	4/15
Pinus strobus	1/9	2/12	2/15	0/15	11/39	1/12	0/3	0/24	1/9	0/12	15/75	3/75
Pseudotsuga menziesii	--	--	--	--	--	5/6	5/9	--	4/6	5/8	9/15	10/14
Pseudotsuga menziesii var. glauca	3/9	10/12	3/9	--	5/27	17/21	5/10	0/9	5/18	22/27	21/73	49/69
Tsuga canadensis	8/9	8/9	--	--	7/18	7/18	9/9	8/18	7/18	4/9	41/54	27/54

* Numerator is number of infections; denominator is number of inoculations.

Canker and dieback was obtained on blue Douglas-fir; only cankers on the other hosts.

The cankers on true fir, spruce, and white pine did not exceed 4.5 cm in length. On the first two hosts the cankers were associated with a very heavy callus and in the case of Norway spruce they were distorted and sunken. Although Phacidiopycnis cankers that occur naturally on white pine are large and covered with erumpent fruiting bodies (Figs. 1, 2), those formed in the tests were comparatively limited. Apparently the conditions that in natural infections result in pronounced cankers were not provided by the experiment. Fruiting of the pathogen on artificial cankers was very meager on white pine and on the true fir. No fruiting occurred on spruce cankers although Phacidiopycnis was isolated from the diseased tissues.

Larger cankers were obtained on blue Douglas-fir and hemlock, generally 4 to 5 cm in length with some measuring 6 to 8 cm. In the former, branches of diameters up to 8 cm became girdled, and dieback resulted. The diseased cortex of hemlock cankers, as on Norway spruce, was not only distorted and sunken but also associated with longitudinal fissuring of the surrounding unaffected bark; check incisions were not accompanied by similar fissuring. Fruiting of the parasite occurred commonly in both Douglas-fir and hemlock cankers. In all instances infections healed over completely in a year's time. The check incisions healed normally.

Spring Series -- The tests were repeated in the spring, in late April and early May. A total of 388 inoculations were made, 162 with American Phacidiopycnis and 226 with Norwegian isolates. These were accompanied by 123 check incisions which healed without infection. Results were comparable with those obtained in the autumn tests. In this spring series, however, approximately twice the amount of infection was obtained on blue Douglas-fir, whereas on true fir, Norway spruce, white pine, and hemlock less infection occurred. In both series approximately the same results were obtained on the other hosts. As in the autumn series, dieback occurred only in blue Douglas-fir.

Tests on Japanese Larch -- The foregoing experiments were supplemented in the early autumn of 1953 and toward the end of spring 1954 with inoculation tests on six vigorous saplings of Japanese larch. The trees were growing in Marsh Garden, Yale University, New Haven, Connecticut. In the autumn, 18 inoculations on growth 1 to 4 years old were made with Phacidiopycnis pseudotsugae isolated from cankered larch and white pine in this country. In early July, 20 inoculations on growth 1 to 5 years old were made with isolates of both American and Norwegian origin. Six check incisions, which healed normally, accompanied each series.

Results of late spring tests in 1954 on actively growing Japanese larch were negative, thus duplicating those from inoculations of the previous summer. In the early October 1953 series, however, two larch trees showed positive results. One tree became infected in one of three inoculations, forming a limited non-girdling canker 2.5 cm long, with pycnidia. A second larch with three inoculations was fully susceptible: two inoculations on branches resulted in girdling cankers and dieback, with abundant pycnidia around the locus of infection; an inoculated terminal about 90 cm in length, which was also girdled and killed, was replaced subsequently by the upright growth of a lateral.

Comparison with Results Obtained in Scotland -- Brief reference is made here to the writer's original positive results from inoculation (21) with Phacidiopycnis pseudotsugae, which have been discussed by Wilson (20). These inoculations were made on the Pacific Coast or typical Douglas-fir considered by the British to be Pseudotsuga douglasii Carr. Pronounced cankering and dieback were obtained experimentally on young trees of this host and at all seasons of the year.

Douglas-fir reacts most favorably to cultural conditions in Great Britain. The species has become adapted readily to the normally mild, moist summers of Scotland and England and to the winters, which as a rule are also correspondingly mild and wet. In Britain, the species is not subjected during the summer to high temperatures and periods of drought as in the United States. These climatic differences probably explain why the author succeeded in obtaining positive results during the summer in Scotland and negative results at that time of year in Connecticut. Climate in Scotland favors not only the growth of young Douglas-fir but also the development of Phacidiopycnis and its attack through wounds made under experimental conditions. Photographs of the writer's summer inoculation results in Scotland are presented here for the first time (Figs. 3, 4, 5).

FIGURE 3. Branch canker on typical Douglas-fir, about 3/4 natural size, resulting from an early summer inoculation with Phaciodiopycnis in Scotland. Weakened terminal, almost completely girdled, died slowly.

FIGURE 4. Girdling canker of a branch terminal, about 3/4 natural size, obtained as in Figure 3. Pronounced swellings occurred at the bases of the main axis and laterals above the region of infection. When collected after 3 years the terminal was still green.

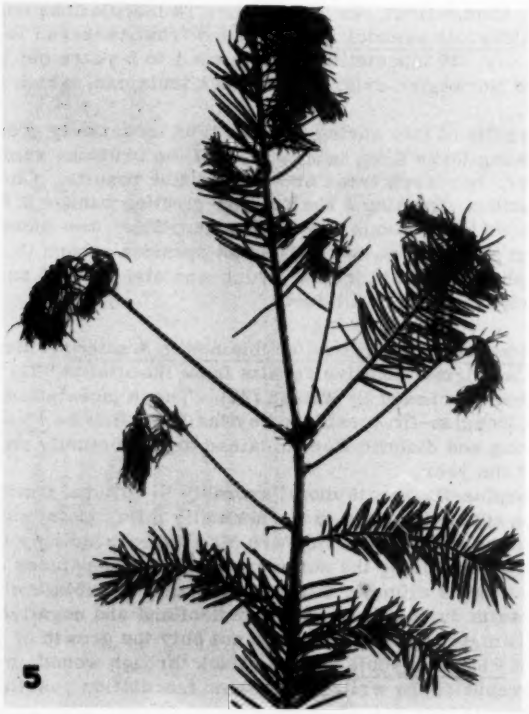


FIGURE 5. Dieback, about 1/4 natural size, obtained as in Figures 3 and 4. When photographed after a month, parts beyond the inoculation site were defoliating and desiccating. The current growth was typically recurved in a "fish-hook" formation.

DAMAGE AND CONTROL

In plantations of Douglas-fir in Europe, the Phacidiopycnis disease may be fatal if the leader is killed back for some distance, particularly where girdling occurs on the main axis of young trees less than 8 feet in height. If this takes place near the ground line, the entire sapling is killed. Cankers on small trees in which the bark is killed more than half way round the stem have practically the same effect as girdling. If the sapling survives the death of its terminal but subsequent fungus attacks involve laterals in the straightening-out process, then a bushy growth results in poor trunk formation (22). Actual loss of trees in young plantations is a matter of concern, for considerable gaps may be left in the final stand if wide spacing has been practiced.

Cankering of older Douglas-fir is not serious. However, damage may be caused to the quality of the wood (23). This is particularly true of canker damage to older stock of Japanese larch in Holland; trees are not killed, but Phacidiopycnis cankers cause large scars on the lower portion of the bole, which later reduces the quality of the lumber (4).

After discovery of the Phacidiopycnis disease, Boyce (3) made a survey of it in Great Britain for the United States Department of Agriculture. He reported that severe damage had occurred on certain planting sites soon after Douglas-fir had been set out. Most damage was seen in a plantation of 10- to 11-year-old Douglas-fir in Scotland, which was reduced to approximately 50 percent of its original stocking. However, he did not find that this amount of damage applied generally.

In foreign countries, environmental factors play an important role in the incidence of Phacidiopycnis disease in young plantations of exotics. Poor planting sites are conducive to attack by the fungus. Frost damage also is a factor in the disease, as Birch (1) demonstrated experimentally in New Zealand. In plantations of Monterey pine (Pinus radiata D. Don), bishop pine (P. muricata D. Don), and Canary Island pine (P. canariensis C. Smith) he showed that infection tied in very definitely with unseasonable frost damage that occurs in the late winter and early spring. At this time, growth of the frost-sensitive species had commenced and infection took place in living but weakened tissue cracked by frost or damaged by snow. Birch reported that infection of this sort rarely occurred on trees beyond the age of 10 years.

The relationship between frost injury and Phacidiopycnis attack has been a subject of controversy (5). Though the fungus very definitely is able to infect and become pathogenic in living tissues weakened by frost, it should not be regarded merely as a saprophyte that occupies dead bark killed by low temperatures (4, 16, 20). As the writer stated previously, desiccated and dead tissues of exotic Douglas-fir resulting from and extending beyond the Phacidiopycnis infection site are colonized not by the extension of growth of the pathogen itself but rather by secondary saprophytes.

In the northeastern United States the Phacidiopycnis disease, which occurs on sapling eastern white pine, presents a problem very different from the one in plantations of exotics in foreign countries. Although the disease is responsible for the death of young trees by girdling, its occurrence is sporadic in naturally regenerated white pine stands. Damage in plantations results from bark cankers on the trunk. These originate in wounds of all types, particularly those resulting from improper pruning methods. Infection through pruning wounds on plantation stock growing on comparatively poor sites may cause serious damage. Hansbrough and Littlefield[3] reported this in a carelessly pruned 25-year-old white pine plantation growing on a sand-blow site in New York State. Wounds healed slowly, particularly those on the less thrifty pines in the stand. As a result, large girdling Phacidiopycnis bark cankers on the trunks of trees in the intermediate and overtopped crown classes contributed to the death of stock of this type.

Ample investigation has shown that Phacidiopycnis pseudotsugae is able to attack and kill living tissue of susceptible conifers but that wounds are necessary for infection. Moreover, Dutch experiments demonstrated that wounding of trunks of Japanese larch is dangerous during the winter when resistance of the tree to the pathogen is lowered. Attack of the fungus is checked by the activity of the trees during their growing season, however (16).

It is evident, therefore, that in preventing or controlling the disease in plantations in the

---

[3]Hansbrough, J. R., and E. W. Littlefield. Phomopsis canker associated with pruning scars on white pine. (Unpublished manuscript in the files of the Northeastern Forest Experiment Station, Forest Service, New Haven, Connecticut) 10 pp., illus. 1948.

Northeast, pruning should be done carefully. Cuts should be clean, leaving no stubs; and the pruning should be done at a time when healing of the cuts will take place promptly. As Connecticut experimentation has shown, there are no hazards of wound infection during the summer. In thinning, extreme care should be taken to prevent wounds or grazed bark on residual trees, such as might result from careless felling or accidental saw or ax cuts. Moreover, plantations should be established on good sites with adequate spacing and should be maintained under conditions most favorable for tree growth. In natural reproduction, the same care should be exercised during the pruning of young trees and the thinning process, even though danger of infection is less in forest stands.

SUMMARY

The canker-dieback disease of Douglas-fir, larch, and other conifers, formerly known as the so-called Phomopsis disease, is caused by a wound parasite. The causative agent should now be known as Phacidiopycnis pseudotsugae (M. Wils.) Hahn. The pathogenicity of the fungus originally was proved by the writer with inoculation experiments in Scotland; his results were confirmed subsequently by independent pathological investigations conducted in Holland.

The perfect stage of Phacidiopycnis pseudotsugae is Phacidiella coniferarum Hahn. Despite world-wide distribution of the Phacidiopycnis stage, the complete life cycle of the pathogen has been found only in the northeastern United States on eastern white pine. Despite the possibility of its being a native parasite, its exact origin is questionable.

The symptoms of the Phacidiopycnis disease on different conifers under conditions of natural infection are described. These refer particularly to the disease on exotic Douglas-fir in Europe.

In the United States, the disease is known only in the Northeast. It is now identified with the so-called "Phoma disease" of white pine saplings, a canker disease recorded in white-pine blister rust literature. Phacidiopycnis has not been found in native Douglas-fir stands.

Positive inoculation results with Phacidiopycnis pseudotsugae are described on young plantation stock of eight species of conifers under American (Connecticut) conditions. The test trees included Abies fraseri, Larix leptolepis, Picea abies, P. glauca, P. pungens, Pinus strobus, Pseudotsuga menziesii and P. menziesii var. glauca, and Tsuga canadensis. Infection was obtained with pure isolates of the fungus collected in the northeastern United States and Norway. It occurred during the autumn and early spring but not during the summer. Reference is made to the writer's foreign inoculation studies with P. pseudotsugae, in which infection tests of young Douglas-fir did succeed under summer conditions in Scotland. Illustrations of the latter are given for the first time.

Damage due to Phacidiopycnis on plantation trees and natural reproduction is discussed. Procedures to be followed in effecting control in the Northeast are outlined.

Literature Cited

1. BIRCH, T. T. C. 1935. A Phomopsis disease of conifers in New Zealand. New Zealand State Forest Serv. Bul. 7. 30 pp.
2. BOYCE, J. S. 1922. A menace to Douglas fir and hemlock. Timberman 23(9): 64.
3. BOYCE, J. S. 1927. Observations on forest pathology in Great Britain and Denmark. Phytopathology 17: 1-18.
4. BOYCE, J. S. 1950. Notes on tree diseases in western Europe. Yale Univ. and U. S. Dept. Agr., Bur. Plant Industry, Soils and Agr. Eng. (mimeo.). 64 pp.
5. DAY, W. R., and T. R. PEACE. 1934. The experimental production and the diagnosis of frost injury on forest trees. Oxford Forestry Memoirs No. 16. 60 pp.
6. GRAVES, A. H. 1914. A preliminary note on a new bark disease of the white pine. Mycologia 6: 84-87.
7. HAHN, GLENN G. 1928. Phomopsis conorum (Sacc.) Died. -- an old fungus of the Douglas fir and other conifers. British Mycol. Soc. Trans. 13: 278-286.
8. HAHN, GLENN G. 1930. Life-history studies of the species

of Phomopsis occurring on conifers. Part. I. British Mycol. Soc. Trans. 15: 32-93.

9. HAHN, GLENN G. 1957. A new species of Phacidiella causing the so-called Phomopsis disease of conifers. Mycologia 49: 226-239.
10. KONING, H. C. 1943. Phomopsis-kanker van Larix en Douglas. Ned. Boschb. Tijdschr. 16(3): 129-137.
11. LAGERBERG, T. 1934. Phomopsis pseudotsugae Wilson, en i Sverige obeaktad Barrträdsparasit. Svenska Skogsvårdsför. Tidskr. 32 (1-2): 71-86. [English summary].
12. MARTIN, J. F., A. E. STENE, and R. A. SHEALS. 1920. How to distinguish and combat the white pine blister rust. Rhode Island State Brd. Agr. Bul. 1. 38 pp.
13. METCALF, HAVEN. 1924. Observations on the Douglas fir canker (Phomopsis pseudotsugae Wilson) in Great Britain. Phytopathology 14: 52.
14. ROBAK, HÅKON. 1952. Phomopsis pseudotsugae Wilson -- Discula pinicola (Naumov) Petr. as a saprophyte on coniferous wood. Sydowia 6: 378-382.
15. SPAULDING, PERLEY. 1956. Diseases of North American forest trees planted abroad. U. S. Dept. Agr., Agr. Handbook 100. 144 pp.
16. VAN VLOTEN, H. 1952. Evidence of host-parasite relations by experiments with Phomopsis pseudotsugae Wilson. Scot. Forestry 6 (2): 38-46.
17. WILSON, MALCOLM. 1920. A new species of Phomopsis parasitic on the Douglas fir. Bot. Soc. Edin. Trans. 28: 47-49.
18. WILSON, MALCOLM. 1925. The Phomopsis diseases of conifers. Great Britian Forestry Comm. Bul. 6. 34 pp. [London].
19. WILSON, MALCOLM. 1930. The Phomopsis disease of cedars. Gard. Chron. 88(2290): 412-413. [London].
20. WILSON, MALCOLM. 1937. The Phomopsis diseases of conifers. Roy. Scot. Forestry Soc. Trans. 51(1): 39-45.
21. WILSON, MALCOLM, and G. G. HAHN. 1929. The history and distribution of Phomopsis pseudotsugae in Europe. Phytopathology 19: 979-992.
22. ZYCHA, H. 1952. Die Phomopsis-Krankheit von Douglasie und Japanlärche. Forstw. Cb. 71(3-4): 65-79.
23. ZYCHA, H. 1953. Die Phomopsis-Krankheit an Douglasie und Japanlärche. Flugbl. biol. Bundesanst. Braunschw. M 15. 6 pp.

NORTHWESTERN FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, NEW HAVEN, CONNECTICUT

RECORDED DUTCH ELM DISEASE DISTRIBUTION IN NORTH AMERICA AS OF 1956[1]

Francis W. Holmes[2]

Several changes in our knowledge of Dutch elm disease distribution now make desirable a revision of last year's map (1).

During 1956, cases of this disease were found in western and southeastern Virginia, as well as at new locations in Delaware, Maryland, West Virginia, and Kentucky. For the first time, cultures of Ceratocystis ulmi (Buisman) Moreau were obtained from elms in Wisconsin,

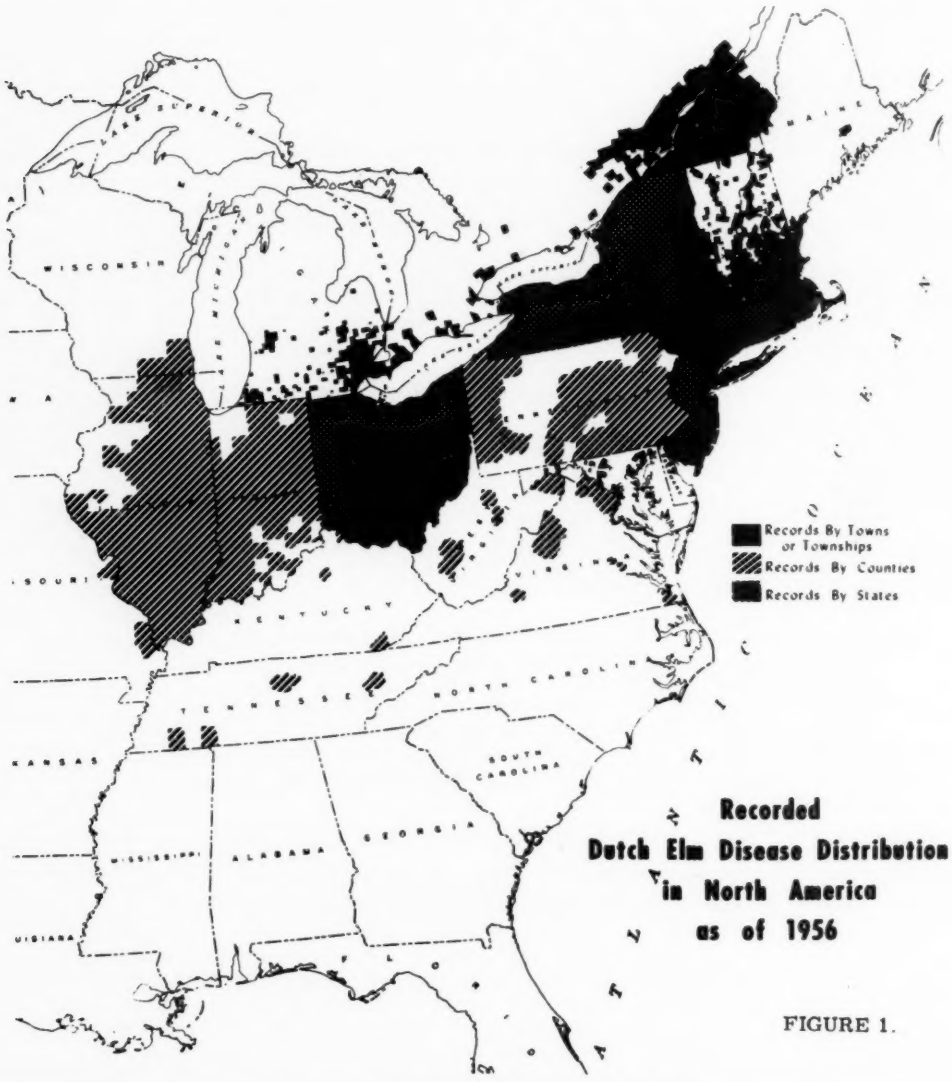


FIGURE 1.

[1]Contribution No. 1105 of the University of Massachusetts, College of Agriculture, Experiment Station, Amherst, Massachusetts.

[2]Assistant Professor, Shade Tree Laboratories, University of Massachusetts.

where 63 cases of the Dutch elm disease were discovered in the southeasternmost six counties (2). Many additional locations were established also in Illinois and Michigan, but the disease has not yet been found in Iowa and no further spread has been recorded in Missouri.

The two major areas of Dutch elm disease infestation in the United States have now nearly merged in Pennsylvania. They may be considered also to have merged in New York, where disease locations are so thoroughly scattered that, for purposes of control measures, pathologists there assume the disease to occur throughout the State. The small outbreak near Orono, Maine has enlarged, and many additional town records have been reported for southern Maine, New Hampshire, and Vermont. A single diseased elm has been found for the first time on the island of Martha's Vineyard, Massachusetts, off Cape Cod.

Last year's map, showing distribution established through 1955, contained the error that in Ontario county data were marked in solid black as though they were town or township data. This year, township data have become available for both Ontario and Quebec. An early report (3) of a case of Dutch elm disease in Norfolk, Virginia, omitted last year, is included in the present map.

The Dutch elm disease has not yet been found in New Brunswick, North Carolina, Georgia, Alabama, Mississippi, Arkansas, Iowa, or Minnesota, all of which adjoin infested States or Provinces. No further cases have been found in Colorado, which is again omitted from this map to permit the use of a larger scale.

Literature Cited

1. HOLMES, F. W. 1956. Recorded Dutch elm disease distribution in North America as of 1955. Plant Dis. Reptr. 40: 351-352.
2. WISCONSIN DEPARTMENT OF AGRICULTURE, DIVISION OF PLANT INDUSTRY. 1956. The Dutch elm disease control program in Wisconsin -- 1956. pp. 1-11. Mimeographed.
3. BEATTIE, R. K. 1934. Dutch elm disease found at Norfolk, Virginia. Plant Dis. Reptr. 18: 191.

UNIVERSITY OF MASSACHUSETTS

THE CURRENT STATUS OF DUTCH ELM DISEASE IN ILLINOIS

R. J. Campana and J. C. Carter

Dutch elm disease (Ceratocystis ulmi) continued to spread into new areas of northern and western Illinois during 1955 and 1956 (Fig. 1), and increased substantially in areas previously affected[1]. From 1950 to 1954 at least 2599 cases were known to occur at 111 localities in 55 counties (Table 1). Since 1954 an accurate tally of diseased trees throughout the State has not been possible. However, from culture records, field diagnoses, and surveys of the most badly affected areas, it was estimated that at least 5,000 new cases of the disease occurred at 155 localities in 75 counties in 1955, and at least 25,000 new cases occurred at 262 localities in 86 counties in 1956.

Presence of the disease was confirmed by laboratory culture in 1955 at 71 new localities, 28 of which were in 20 counties previously unreported (Table 2). In 1956 the disease was confirmed at 97 new localities, 19 of which were in 11 previously unreported counties (Table 3). During the two-year period the disease spread northward to the Wisconsin boundary in Lake, McHenry, Boone, and Winnebago Counties. Westward movement of the disease front occurred at Winnebago in Winnebago, Oregon in Ogle, Dixon in Lee, and Rock Falls in Whiteside Counties in the Rock River Valley, and at Peoria in Peoria County.

In 1955 isolated infections of the disease occurred at Rockford in Winnebago, at Galesburg in Knox, and at Rushville in Schuyler Counties (Fig. 1). In 1956 an isolated case was discovered at Quincy in Adams County. No additional diseased trees were reported in 1956 from the isolated areas at Rushville and Galesburg, nor was the disease found in surrounding counties.

Of special interest in 1956 was the spread of the disease beyond Illinois into Wisconsin[2] at 14 localities in 6 counties, and its close approach to Iowa. It is anticipated that all of the 16 remaining Illinois counties where Dutch elm disease has not been found will have the disease by 1957. Spread of the disease beyond Illinois into Iowa during the coming season is a serious probability.

In 1954 the Dutch elm disease had been found in 40 newly affected counties in Illinois[3]. Its known spread to only 20 more newly affected counties in 1955 and 17 (11 in Illinois and 6 in Wisconsin) in 1956, aside from possible adverse climatic influences, is attributed primarily to absence of the phloem necrosis virus disease in the areas affected. In most of the 40 counties invaded by Dutch elm disease in 1954 phloem necrosis had weakened or killed thousands of trees prior to the arrival of Dutch elm disease. The bark beetle populations in some places in these counties had reached extremely high proportions compared to those in areas with no phloem necrosis, because of the tremendous volume of elm material available for breeding in elms dead and dying of phloem necrosis.

Another possible contributing factor to the difference in spread of the disease between 1954 and the following 2 years was the effect of the severe drouth years of 1953 and 1954. Southern counties of Illinois, where most spread of Dutch elm disease occurred in 1954, were generally more adversely affected by drouth than the northern counties. It was observed that weakening of elms by drouth followed by subsequent invasion by bark beetles alone caused the death of thousands of elms in southern Illinois during 1954. Chances for greater spread of the disease through more beetle activity were increased tremendously.

Of greater immediate significance than extension of the disease to additional counties was its spread and development in counties previously affected because of the large numbers of trees affected at single localities. In the Chicago area alone the disease was confirmed in 101 communities for the first time. Development of the disease at epiphytotic levels occurred for the first time at Bloomington in 1955 with 240 new cases; at Rockford in 1956 with more than 200 new cases; and in the Fox River Valley from Aurora to Dundee with several hundred new cases in 1956.

Complete surveys were made in Bloomington and Champaign in studies of the incidence of Dutch elm disease following phloem necrosis. In Bloomington the Dutch elm disease increased from 10 cases in 1954, to 242 in 1955 and to 507 in 1956. Phloem necrosis in Bloomington

---

[1]Campana, R. J., and J. C. Carter. Spread of Dutch elm disease in Illinois in 1954. Plant Dis. Reptr. 39:245-248. 1955.

[2]Anonymous. The Dutch elm disease control program in Wisconsin. Wisconsin Dept. Agr. Mimeo Report, Dec., 1956. Madison, Wis.

[3]Campana and Carter, see footnote 1.

Table 1. Record of Dutch elm disease in Illinois

Year	Counties Affected	Localities Affected	Trees Affected
1950	1	1	1
1951	4	6	11
1952	9	14	25
1953	15	34	495
1954	55	111	2,067
1955	75	155	5,000[a]
1956	86	262	25,000[a]

[a]Estimated

Table 2. Counties newly affected with Dutch elm disease in 1955.

County	Localities Affected	Trees Affected
Cumberland	1	1
DuPage	6	18
Grundy	1	4
Kendall	1	1
Knox	1	1
LaSalle	3	3
Logan	1	1
Macoupin	1	1
Marshall	1	1
Menard	1	1
McHenry	1	1
Perry	1	1
Putnam	1	1
Randolph	1	1
Scott	1	1
Schuyler	1	1
Tazewell	2	2
Union	1	1
Washington	1	1
Williamson	1	1
Totals	28	43

Table 3. Counties newly affected with Dutch elm disease in 1956

County	Localities Affected	Numbers of Cases
Adams	1	1
Boone	3	9
DeKalb	2	5
Hardin	1	1
Lee	2	3
Monroe	2	2
Ogle	4	10
Peoria	1	3
Sangamon	1	1
Wabash	1	1
Whiteside	1	1
Total	19	37

Table 4. Incidence of Dutch elm disease and elm phloem necrosis in Champaign-Urbana

Year	Trees Affected		Percent Dutch Elm Disease
	Phloem Necrosis	Dutch Elm Disease	
1944-50	428	0	0.0
1951	359	1	0.3
1952	555	11	2.0
1953	388	164	30.0
1954	179	694	80.0
1955	123	1805	94.0
1956	60	1836	97.0
Total	2092	4511	

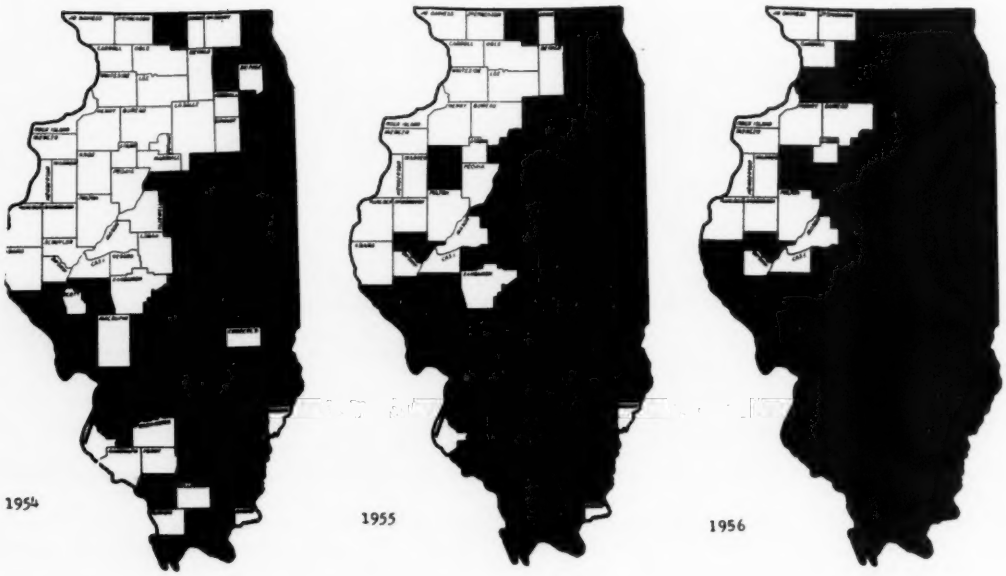


FIGURE 1. Known distribution of Dutch elm disease in Illinois from 1954 to 1956, indicating spread each year by counties.

decreased from 400 cases in 1954 to 200 cases in each of the years 1955 and 1956. In Champaign-Urbana the percentage of elm loss from Dutch elm disease has increased steadily and rapidly from 0.3 in 1951, to 94.0 in 1955 and 97.0 in 1956, whereas the percentage loss from phloem necrosis appears to have declined proportionately (Table 4).

It is not known exactly how many trees were affected by both diseases since positive diagnoses by detailed examinations of doubtful trees for phloem necrosis and by laboratory culture for Dutch elm disease were not made for all cases. Where detailed diagnoses of trees in question were made for both diseases, positive symptoms of phloem necrosis were observed in less than 5 percent of trees with evidence of Dutch elm disease. However, the relative slowness of both internal and external symptoms of phloem necrosis to develop, the predisposition of even symptomless trees weakened by early stages of phloem necrosis to invasion by bark beetle carriers of the Dutch elm disease fungus, and the greater speed and certainty of development of Dutch elm disease suggest that phloem necrosis may have been present but undetected in some cases. This is further indicated by the apparent but rapid decline of the incidence of phloem necrosis coincident with the spread of Dutch elm disease, with a substantial residual elm population remaining. Where phloem necrosis has run its course in other communities in Illinois in the absence of Dutch elm disease, a high loss has been maintained annually in most instances until the residual elm population has been reduced well below 50 percent of the original elms. That this has not happened in Champaign-Urbana is indicated by the data in Table 4.

These observations suggest that where Dutch elm disease follows phloem necrosis, it not only exceeds phloem necrosis in the rate of its increase, but obscures the presence of phloem necrosis. The result is to make uncertain the separate influence of phloem necrosis in reduction of the elm population affected by both diseases.

ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA, ILLINOIS

VASCULAR WILT OF MIMOSA IN FLORIDA

George F. Weber[1]

The wilt disease of mimosa, Albizzia julibrissin, observed in Florida in 1952, has been spreading since that time. Recently a wilted tree was observed in Gainesville showing the following symptoms correlated with the time of year. The specific tree in question calipered approximately 10 inches DBH and was 12 years old, growing luxuriantly in a lawn.

The first symptoms of the disease, manifested by a slight drooping and wilting of the foliage, appeared on May 1 on a single branch, and was followed within the next 2 days by a slight yellowing of the leaflets at the extremity of the branch. The tree at this time had fully mature foliage and was rapidly approaching blossoming time. The first flowers opened on May 10. On May 15, several larger branches showed the wilt symptoms (Fig. 1) and the yellow foliage, and the portions of the tree that showed the first symptoms exhibited considerable shedding (Fig. 2) of the leaflets, leaving the rachis attached. At this time there appeared to be many erupting, linear trunk lesions which were from 3 to 10 inches long and from which sap exuded and formed streaks down the trunk (Fig. 3). This sap flow was attractive to insects.

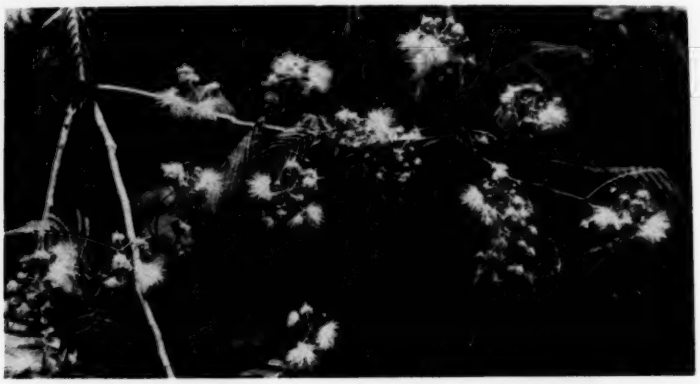


FIGURE 1. Flowering branch of Albizzia julibrissin showing drooping and wilting of the foliage, which are the first symptoms of the disease.

FIGURE 2. A tree showing defoliated top branches and healthy branches toward the right and at the bottom.

[1] Professor of Plant Pathology, College of Agriculture, University of Florida.

FIGURE 3. Portion of trunk of diseased tree, showing exuding sap as a symptom of the disease.

FIGURE 4. Inoculation test showing four inoculated seedlings dying from the disease at left and two healthy checks at right.

The tree became rapidly involved in the disease, one branch after another in close succession showing similar conditions, and by June 1 several branches were entirely defoliated, and the foliage on all remaining parts of the tree showed symptoms.

On July 1, the tree had become entirely defoliated and was dead. It was removed and cross and longitudinal sections of the roots and trunk were studied. The wood was darkened in the areas involving the xylem and the last 1 and often 2 annual layers. This darkened area continued longitudinally into the trunk and all of the larger branches. The fungus was isolated from roots, branches and twigs by planting infected host tissue on potato-dextrose agar hard plates. It grew rapidly in culture and compared favorably with descriptions of Fusarium oxysporum f. perniciosium (Hept.) Toole. The fungus remained viable in culture dishes on the laboratory shelf for more than 6 months.

The source of the inoculum is not known and theorizing is highly conjectural inasmuch as the nearest diseased specimens have been at least 100 miles away. This tree was grown from seed obtained from a distant area where the disease has not been seen. The organism in culture was used as inoculum for testing its pathogenicity on seedlings. One-year-old potted plants were inoculated by stabbing the soil in flower pots containing them, and a spore suspension introduced into these areas. Seedlings inoculated in this fashion developed the disease typically, and after 3 weeks had become entirely defoliated, whereas the check plants were free from the disease (Fig. 4). An interesting symptom, probably not previously recorded, was the extensive bleeding that took place on the trunk of the tree in the early stages of the disease. Possibly this condition was aggravated by the fact that the tree was approaching the critical blooming stage at this time.

There is every reason to believe that this disease is identical with the one described by Hepting (1) and Toole (2). The interesting thing about the appearance of the disease on isolated trees in Gainesville, Florida, is the distance from this isolated location to the nearest hitherto reported area of the disease. The Albizzia spp. are extensively grown as ornamentals in this State, and the spread of the disease in this area would be devastating in view of the current popularity of this conspicuous flowering, rapidly growing tree.

Literature Cited

1. HEPTING, GEORGE H. 1939. A vascular wilt of the mimosa tree, Albizzia julibrissin. U. S. Dept. Agr. Circ. 535: 1-10.
2. TOOLE, E. R. 1941. Fusarium wilt of the mimosa tree, Albizzia julibrissin. Phytopathology 31: 599-616.

DEPARTMENT OF PLANT PATHOLOGY, COLLEGE OF AGRICULTURE, UNIVERSITY OF FLORIDA, GAINESVILLE, FLORIDA

NEW OR UNUSUAL PLANT DISEASES IN LOUISIANA

A. G. Plakidas[1]

I. TIMBER ROT OF TOMATOES

On April 5, 1957, several tomato plants in a test plot at the Plaquemines Parish Experiment Station, Port Sulphur, Louisiana were observed dead or dying with a sudden type of wilt. Affected plants were scattered through the field, not localized in any one spot. In every case, there was a dead portion of the stem, brown in color and about 2 to 3 inches in length, usually close to the soil surface. The plants had been set in the field February 28, had reached a height of about 18 inches, and had already been tied to stakes.

Specimens of affected stems were taken to the laboratory and placed in a moist chamber. Within 24 hours, profuse white mycelium grew from the tissues (Fig. 1) and sclerotia of Sclerotinia sclerotiorum formed in the pith of the stems in about 3 days. The fungus was also obtained in pure culture.

FIGURE 1. Tuber rot of tomato. Growth of Sclerotinia sclerotiorum on affected stems.

While Sclerotinia stem rot of tomato is not a new disease (several references of its occurrence are found in pathological literature) no record of its previous occurrence in Louisiana has been found in our files. Weather conditions were particularly favorable for the development of the disease. Heavy and prolonged rains fell in March, often flooding parts of this planting, and temperatures were cooler than normal for this region.

II. ENTOMOSPORIUM LEAF SPOT OF PHOTINIA GLABRA

Photinia glabra (Thunb.) Maxim. is a popular ornamental shrub in Louisiana. It is propated extensively by many nurseries for the reason that there is a ready market for it and also because, until now at least, it has been free of any serious disease or insect pests and has been considered a "fool-proof" plant to grow.

On April 11, 1957 the writer was called to examine some photinia plants in a nursery in Folsom, Louisiana, that had been so badly defoliated by some disease the previous summer as to be rendered unsalable. These plants, comprising a solid plot of about one acre, were found affected with very numerous purplish to brown, mostly circular, necrotic spots on all their

[1] Professor of Botany and Plant Pathologist, Louisiana State University, Baton Rouge, Louisiana.

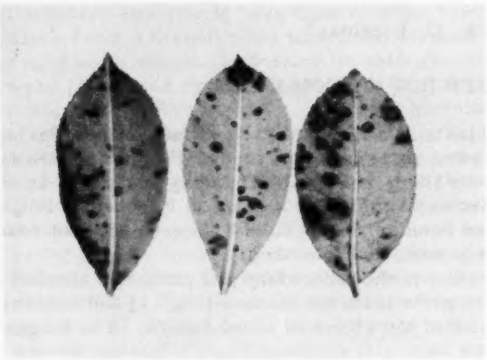


FIGURE 2.
Entomosporium spots on Photinia glabra leaves. Approx. 3/4 natural size.

leaves (Fig. 2). The plants had been pruned back severely in late summer, and the new growth that developed after pruning was also covered with spots, indicating that infection had continued to spread during fall. Leaf specimens were collected and taken to the laboratory for examination. Acervuli bearing conidia of Entomosporium maculatum were present in great abundance on the lesions.

On May 9, P. glabra plants in several other nurseries as well as in home yards in the Folsom area were examined, but the disease was not found. Inquiries were made concerning the source of the plants of the nursery in which the disease occurred, and it was learned that these plants originated as cuttings from some large plants growing in the yard of the homestead of an older nursery in the neighborhood which had since been sold. Since it was known to the writer that several pear trees severely affected with Entomosporium leaf spot were growing in this yard in close proximity to the photinias, it was suspected that the inoculum might have come from the pears. However, when the place was visited, most of the photinias were found free of disease. The only exception was three plants growing directly under a pear tree, the lower branches of the pear interlacing with some of the photinia branches. The pear foliage was severely spotted; a limited number of spots were present on the photinia leaves, but it is not certain that these spots were caused by Entomosporium because no fruiting bodies of the fungus were found on them. A small loquat tree (Eriobotrya japonica) growing close to the pear trees had foliage covered with necrotic brown spots; acervuli and conidia of Entomosporium occurred abundantly on these lesions.

So far as the writer has been able to ascertain, this constitutes the first report of Entomosporium maculatum on Photinia glabra, although the disease is common on another species of photinia, P. arbutifolia (Christmasberry) in California. Entomosporium maculatum Lév. (Fabraea maculata Atk.) has been reported on many members of Rosaceae (pear, quince, crabapple, hawthorn, serviceberry, cotoneaster, mountain ash, medlar, Christmasberry, loquat, pyracantha), but, with the possible exception of pear and quince, it has not been determined with certainty whether or not the fungus from one of these hosts will infect another. Circumstantial evidence, at least, points to the existence of specialized host races. For example, Smith[2] states in reference to the disease on Christmasberry: "F. maculata is the cause of a common leaf and fruit spot of the pear in the eastern parts of the country but is not known on this host in California. The fungus may not be identical in the two hosts."

The source of infection of Photinia glabra in Louisiana has not been determined. The pear is suspected even though observational evidence does not support this suspicion. The disease on pear has been in Louisiana with certainty since 1930, when it was first observed, and probably longer. Pear trees (all with leaf spot) occur widely in home yards in the vicinity of the commercial nurseries in the Folsom area, and if the fungus from pear were pathogenic to photinia, infection of photinias in nurseries would have occurred long ago, yet the disease appeared for the first time last summer and up to this date appears to be confined to one nursery. At the Fruit and Truck Experiment Station in Hammond, Louisiana, there is a planting

[2] Smith, R. E. 1940. Diseases of flowers and other ornamentals. California Agr. Ext. Service Circ. 118, p. 53.

of photinias within about 500 yards of a group of several pear trees, all badly diseased with leaf spot, and no trace of the disease on the photinias.

It is conceivable that a mutant of the pear fungus arose that is pathogenic to photinia. This, admittedly, is mere speculation. Cross-inoculation experiments are in progress in the hope of obtaining more definite information.

DEPARTMENT OF BOTANY, BACTERIOLOGY AND PLANT PATHOLOGY, LOUISIANA STATE UNIVERSITY, BATON ROUGE, LOUISIANA

DISEASES OF TEMPERATE CLIMATE CROPS IN BOLIVIA

Frank H. Bell and Segundo Alandia B.[1]

Bolivia is one of the two inland countries in South America. This relative isolation, the large proportion (about 65 percent) of Indian population, the past emphasis on mining, and the difficulty of transportation in much of the country have been important factors in the general lack of technical agricultural knowledge and modern agricultural practices. The landowner generally lives in the bigger cities, and leaves much of the farming operations to the Indian or mestizo laborers and managers, who farm much as they have for decades. The relatively specialized knowledge of plant diseases is even more lacking. The first agronomy school in the country, and therefore the first course in plant pathology, was established in Cochabamba about fifteen years ago. Until the last two or three years, about the only regularly conducted disease control programs were those carried out by some farmers to control the mildews of grape and Coryneum blight and powdery mildew of peach.

Bolivia has an area of about 415,000 square miles, or about the same as all of the States east of the Mississippi and north of the Ohio River and the Mason-Dixon line. Geographically, and therefore agriculturally, the country can be divided into three principal areas: the Altiplano, the Valleys, and the Eastern Lowlands or Oriente. The Altiplano occupies the whole length of Bolivia on the western side. Bordered by the western and eastern ranges of the Andes, it is an unhospitable, treeless, mostly level area with an altitude of about 12,500 feet. In spite of the rigorous climate, and the fact that it comprises about 14 percent of the total area of the country, 57 percent of the total Bolivian population of about 3,500,000 live there. Principal crops in this area are potatoes, barley, broad beans, quinoa (Chenopodium quinoa), and oca (Oxalis tuberosa). Frost may occur at any time of the year in the Altiplano.

The Valley Section includes the area between the Altiplano and the flat eastern Lowlands, and reaches a width of 250 miles. Although some areas between the valleys approach Altiplano conditions, much of the area lies between an altitude of 6,000 and 10,000 feet. Temperate climate crops such as wheat, corn, potatoes, grapes, peaches, and a wide range of vegetables predominate. In this area, light frosts occur in the winter.

Because most of the Bolivian population lives in the mountains, and because the country is world-known for its tin, it is not generally realized that 70 percent of the country is tropical and semi-tropical lowlands. Rubber, Brazil nuts, cacao, rice, and citrus are crops in the isolated northern area of the Oriente. Much of the hopes for the agricultural future of the country are being placed in the Santa Cruz region, which is near the center of the country. A railroad connects it with Brazil, another one is being constructed from Argentina, and a modern paved road is being completed from Cochabamba, where products are shipped by rail to the populous Altiplano. Main crops in the Santa Cruz area are sugar cane, rice, citrus, corn, yuca, and coffee, and vegetables in the winter season.

Practically a separate region is the Yungas, an area lying north of La Paz on the eastern slopes of the Andes at an elevation of 4,500 to 6,000 feet. Here important crops are coca (which yields cocaine), citrus, and bananas.

Until after the Second World War there were no agricultural experiment stations in Bolivia. There are now four, all under the Servicio Agrícola Interamericano, a cooperative project between the United States and Bolivian Governments. One is located on the shores of Lake Titicaca, near Achicachi (altitude 12,500 feet), one in the Cochabamba Valley (altitude 8,500 feet), one north of Santa Cruz (altitude 1,000 feet), and one at Riberalta, in the northern rubber area (altitude 550 feet). Rainfall increases from southwestern to northern Bolivia. In the southern Altiplano it averages 10 to 12 inches, in the northern Altiplano there is from 16 to 28 inches, at Cochabamba about 22 inches, at Santa Cruz from 50 to 60 inches, and in northern Bolivia it reaches 80 inches. Rainfall in the lower slopes of the Andes, where agriculture is practically nil, is much more abundant. There is a winter dry season (April through November) which is most accentuated in the mountains. In this latter area irrigation is always practiced where water is available, as even in the rainy season the precipitation is unpredictable.

[1]The authors acknowledge the assistance of J. A. Stevenson, E. K. Cash, P. L. Lentz, and A. L. Robert, of the Agricultural Research Service of the United States Department of Agriculture, who identified many of the fungi discussed in this report. Present address of the senior author: Av. Estados Unidos 485, Pueblo Libre, Lima, Peru.

Published references to plant diseases in Bolivia have been mostly of a mycological nature. These have been brought together, and many more added as a result of the collections of Dr. Cardenas, by Stevenson and Cardenas in their "Lista Preliminar de los Hongos de Bolivia" (1). However, relatively few of the host plants of the fungi listed are of agricultural importance. During the nearly four years that the authors worked together as Plant Pathologists in the Servicio Agrícola Interamericano, plant diseases have been observed and collected in most of the principal agricultural regions of Bolivia. As a result, the number of plant disease causal organisms are now about triple those previously known.

CEREALS

Wheat stem rust (Puccinia graminis tritici) can be destructive below an altitude of about 9,500 feet. Vallega and Coronel, working in Argentina with material from Bolivia, determined that races 14, 15, 17, and 11 are present in the country. At the Cochabamba Experiment Station it has been determined that race 15B is prevalent. Puccinia glumarum is destructive only above an elevation of about 9,000 feet, where it has been a principal limiting factor in the cultivation of wheat. Improved wheat varieties have been introduced into the Cochabamba Valley, and the work of the Servicio Agrícola Interamericano will soon result in improved wheats resistant to stem rust for the valleys, and stripe rust-resistant wheats that will permit wheat culture in the highlands. Septoria tritici and S. nodorum have each been observed to be destructive in a limited area. The smuts (Ustilago tritici and Tilletia sp.) are present, but outside of reported damage in the South, are of little importance.

Covered smut of barley (Ustilago hordei) is often destructive in the Altiplano, and fields with over 50 percent smutted heads have been noted. Ustilago nuda is of much less importance. Puccinia graminis is of little importance, because most of the barley is grown at too high an altitude. Puccinia glumarum has been observed on barley. Scald (Rhynchosporium secalis) is often a destructive disease.

Oats is not a common crop in Bolivia, but its stem rust (Puccinia graminis avenae) was observed to be damaging at an altitude of 11,000 feet as well as at lower altitudes. Puccinia coronata has been collected on this host in the La Paz area (1).

Corn rust (Puccinia sorghi) is common throughout the country, but is generally considered to be of minor importance because it arrives late in the season. Smut (Ustilago maydis) and leaf spots are widespread, but apparently do little damage. In the Santa Cruz area specimens were collected that were infected with Helminthosporium maydis (Cochliobolus heterostrophus), Pseudomonas alboprecipitans, and Curvularia sp. Phyllachora maydis and Cladosporium herbarum have been collected on corn (1). Puccinia purpurea is present on sorghum in the Santa Cruz area.

OTHER FIELD CROPS

Downy mildew (Peronospora trifoliorum), Pseudopeziza medicaginis, and to a lesser extent rust (Uromyces striatus), are prevalent on alfalfa in the Cochabamba Valley. An unidentified crown rot is sometimes troublesome in alfalfa and sweet clover. Uromyces trifolii and U. flectens have been collected on clover (1). Bacterial pustule (Xanthomonas phaseoli) is present on soybean at the Experiment Station near Santa Cruz.

White rust (Albugo tragopogonis) is prevalent on sunflower at the Cochabamba Experiment Station, and powdery mildew (Erysiphe cichoracearum) and rust (Puccinia helianthi) have been collected. A very destructive blight in which large portions of the leaves and finally the whole sunflower plant become black and necrotic has been noted near Santa Cruz, and similar but less virulent symptoms are present at Cochabamba. It appears to be due to a virus which is also known in Argentina.

Alternaria longipes has been found on tobacco. Rust (Melampsora lini) has been collected on flax in the Cochabamba Valley.

VEGETABLES

Tomatoes have many diseases, but the most damaging in the country as a whole are the virus diseases. For example, in a 1955-56 planting at the Cochabamba Experiment Station consisting of 311 plants of some two dozen varieties, a count early in the season revealed that 60 percent of the plants were infected with spotted wilt, and 71 percent with a curly-top type. Only one variety, Rey Humberto, a small fruited type obtained from Argentina, was

free of these viruses at the time, although several plants later succumbed to spotted wilt. As this is by far the most resistant tomato to these viruses, crosses have been made with a large fruited type in hope that the desirable qualities of each will combine. The "curly top" of tomato is apparently different from that of beet, however, as beet curly top was transmitted by the leafhopper, Agallina ensigera, to 13 out of 15 garden beet plants from infected swiss chard, but not one of 15 tomato plants contracted the virus. Mosaic viruses are also common on tomatoes, and others, such as one which causes intense curling and deformation of the leaves, have been noted. Septoria lycopersici sometimes causes damage to tomatoes, especially in the Cochabamba Valley. Powdery mildew (Erysiphe polygoni) is common when tomatoes are grown in the greenhouse of the Cochabamba Station. Often destructive in the sandy soils of the Santa Cruz area is the root-knot nematode (Meloidogyne sp.).

Garden beets, sugar beets, and swiss chard at the Cochabamba Experiment Station are heavily infected with curly top. A leafhopper, Agallina ensigera, is a vector. A few field-collected leafhoppers transmitted the disease to swiss chard in the greenhouse, and several months later individuals of the same species of leafhopper which had fed on two of the swiss chard plants transmitted curly top to 13 out of 15 garden beet seedlings. Sugar beets and garden beets at the Station also are infected with rust, Uromyces betae.

Peas, especially in the Altiplano, are often heavily infected by a leaf and pod spot caused by Ascochyta pisi. In the Department of Chuquisaca Septoria pisi has been found on this host. Lupines are commonly cultivated in the higher altitudes. Phyllosticta ferax was collected on plants growing near Copacabana, and a rust, Maravalia elata, has been collected on it in a few places (1). Rust (Uromyces phaseoli) has been collected on bean. The broad bean is extensively grown in the Altiplano in the summer and in the valleys in the late winter and spring. Chocolate spot (Botrytis sp.) and rust (Uromyces fabae) are common diseases of this legume, the latter being so destructive in the valleys in the late summer that it is a major factor in the absence of the crop at that time of year. A crown and root rot is sometimes destructive to the broad bean, and the predominance of a Fusarium in isolations indicates that this fungus may be responsible. Cabbage and cauliflower have been found infected by Alternaria brassicae (spores up to 170 microns long) and radish and wild crucifers with Albugo candida. Celery is not common, but Septoria sp. has been noted on it.

Onion has been found infected with Sclerotium cepivorum in the Departments of Cochabamba and Chuquisaca, and a downy mildew, Peronospora destructor, is apparently widely distributed in these two areas. Rusts are present on hot pepper in several areas, sometimes to such an extent as to alarm the farmers. Aecidium capsici has been identified, although in one collection teliospores have been found in addition to aeciospores. In Cochabamba, a downy mildew (Phytophthora capsici) has been found on pepper. A species of Colletotrichum causes a leaf spot of hot pepper in the Department of Santa Cruz.

Powdery mildew (Erysiphe cichoracearum) is a destructive disease of many of the cucurbits in the Valleys, and Septoria cucurbitacearum has been collected on melon in Cochabamba (1). Cercospora sp. was found to be damaging a field of lettuce near Santa Cruz. Papa-lisa (Ullucus tuberosus) is a tuber-bearing plant widely cultivated in the higher altitudes. An Aecidium has been found on it in a high valley above Cochabamba; it may be new to the host.

The root-knot nematode is distributed throughout much of the country, but is particularly destructive in the sandy soils of the Santa Cruz area to plants such as tomatoes, carrots, beets, and turnips.

FRUIT

The peach is attacked by several parasites that would ordinarily make regular control measures an economic necessity, but very few farmers carry them out. Perhaps the most destructive is powdery mildew (Sphaerotheca pannosa); in some places it is easy to find peaches with half the surface covered with the white fungus, and damage to young shoots is severe. Coryneum carpophilum blight is also often destructive. Crown gall (Agrobacterium tumefaciens) is widespread, as is brown rot (Monilinia fructicola). Peach rust (Tranzschelia pruni-spinosae) is everywhere, although it probably builds up too late in the season to be very damaging. Leaf curl (Taphrina deformans) is found throughout, but is of little economic importance. Rust and Coryneum blight are also found on plum.

Grape is equal to peach in the necessity of disease control measures. Powdery mildew (Uncinula necator) is especially destructive in the lower Cochabamba Valley, practically destroying the crop in some areas. Downy mildew (Plasmopara viticola) is more prevalent in southern Bolivia, where the country's best wine is produced. Regular spray programs are

sometimes carried out there. In that region crown gall has been found on the aerial branches. Isariopsis clavispora is found in the eastern lowlands, although grapes are not important in this area.

Scab (Venturia inaequalis) is found on apple, and in southern Bolivia crown gall has been found on the aerial branches. Mycosphaerella fragariae has been collected on strawberry.

MISCELLANEOUS

Quinoa is an important and nutritive plant of the highlands. It is often called a cereal, but it belongs to the genus Chenopodium. Powdery mildew (Peronospora effusa) is widespread on this host.

Two rusts (Melampsora larici-populina and M. medusae) have been collected on poplar (1).

Literature Cited

1. STEVENSON, J. A., and M. CÁRDENAS. 1949. Lista Preliminar de los Hongos de Bolivia. Lilloa (Tucumán, Argentina) 21:

ESTACION EXPERIMENTAL AGRICOLA, SERVICIO AGRICOLA INTERAMERICANO, COCHABAMBA, BOLIVIA

NEW RECORDS OF PLANT DISEASE OCCURRENCE

RUST OF ST. AUGUSTINE GRASS IN FLORIDA

By Edwin H. Todd[1]

Rust (presumably Puccinia stenotaphri (Syd.) Cummins[2]) of St. Augustine grass (Stenotaphrum secundatum) was found early in 1957 in relatively shady, moist places in lawns and pastures around the Lake Okeechobee area from Canal Point to Belle Glade, Florida. While no comprehensive evaluation has been made of the rust on the grass here, the disease appears to be fairly prevalent although at the moment actual damage to the grass seems negligible.

P. stenotaphri was originally described by Sydow[3] on S. glabri from Africa in the uredo stage only. The perfect stage of the rust was described by Cummins[4] on S. dimidiatum from Mauritius. Recently, P. stenotaphri was found in Puerto Rico, the first report of it occurring in the New World[5]. Insofar as known, the present report records the first observation of a rust on St. Augustine grass in the continental United States.

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, BELTSVILLE, MARYLAND.

[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Canal Point, Florida.

[2]Acknowledgment for tentatively identifying the rust is given to Mr. John A. Stevenson, Principal Mycologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

[3]Sydow, H. and P. 1909. Ann. Myc. 9:544.

[4]Cummins, G. B. 1945. Bull. Torr. Bot. Club 72:213.

[5]Correspondence from Mr. Stevenson.

NEEDLE-CAST OF SITKA SPRUCE IN OREGON

By John Hunt and Ernest Wright

Plantations of 25-year-old Sitka spruce (Picea sitchensis (Bong.) Carr.) growing at an elevation of 500 feet in the Coast Range of Oregon about 20 miles southeast of Astoria, have in recent years shown considerable dropping of two-year-old needles. The needle-cast fungus responsible has been identified as Lophodermium filiforme Darker (F. P. 91726 Portland). Current year's needles are not infected. On nearby mature native Sitka spruce, the 3- and 4-year-old needles are not infrequently infected with L. piceae (Fuckel) v. Hoehnel (F. P. 91724 Portland). The latter fungus caused very little premature defoliation. It appears that the needle-cast parasite of the planted trees did not spread from nearby native spruce but most probably was brought in on the planting stock. The only previous report of L. filiforme on the Pacific Coast was from Alaska and this appears to be the first report from the Pacific Northwest. Duplicate collections have been deposited at the National Fungus Collections, Beltsville, Maryland.

PACIFIC NORTHWEST FOREST AND RANGE EXPERIMENT STATION, PORTLAND, OREGON

CORRECTIONS

REPORTER for May 15 (Vol. 41, No. 5), page 441: This work was done at the SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, STATE NATURAL HISTORY SURVEY, URBANA, ILLINOIS, instead of the location given.

REPORTER, Vol. 41, No. 6, June 15, 1957, article by T. W. Bretz and W. D. Buchanan, "Oak Wilt Fungus Not Found in Acorns from Diseased Tree." In paragraph 3, line 10, the sentence should read "These embryos remained dormant and no "scar" tissue developed on the injured cotyledons." In other words, insert the word "no" before the word "scar".

IN THIS ISSUE (Continued).

Association of the spread of Dutch elm disease in Illinois and neighboring States with occurrence of phloem necrosis is discussed by R. J. CAMPANA and J. C. CARTER, page 636.

GEORGE F. WEBER describes seasonal progress of symptoms of mimosa vascular wilt in Florida, page 640.

A. G. PLAKIDAS reports Sclerotinia stem rot of tomatoes and Entomosporium leaf spot of Photinia glabra as newly recorded in Louisiana, page 643.

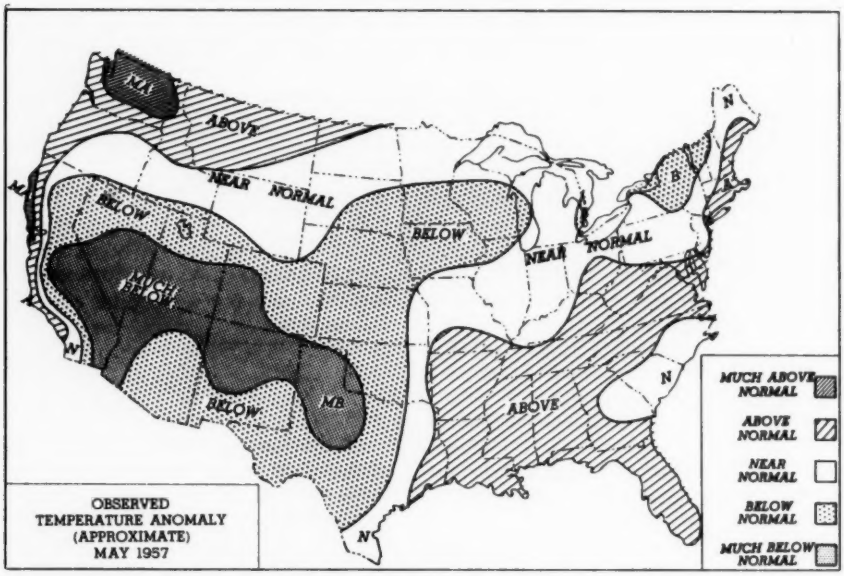
FRANK H. BELL and SEGUNDO ALANDIA B. report diseases of temperate climate crops observed in Bolivia, page 646.

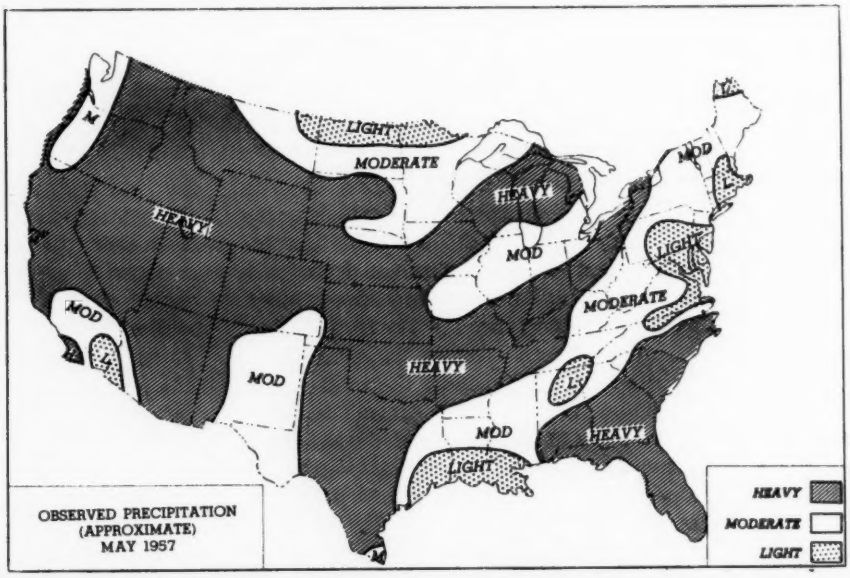
New records, page 650: Rust on St. Augustine grass in Florida, by EDWIN H. TODD. Needle-cast of Sitka spruce in Oregon, by JOHN HUNT and ERNEST WRIGHT.

Corrections, page 650.

May weather, page 652.

Manuscripts for the Reporter, page 653.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and bibliographies. (Note -- only one copy is needed.) Footnotes should be typed at the bottom of the page.

(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

(8) TABLES should be carefully thought out with particular attention to the Reporter's limitations in reproduction. Make titles and headings definite and self-explanatory. Designate footnotes in tables with superscript lower-case letters. Be sure that text discussion agrees with the data in the table. Do not abbreviate names of crop varieties.

(9) REPRINTS cannot be supplied since there is no way in which we can be reimbursed. However,

(10) The MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- for Model 1250. Most of the Experiment Stations have this type of multilith machine.

(11) We are the official correspondents in the United States of the PLANT PROTECTION BULLETIN published monthly by the United Nations Food and Agriculture Organization. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our monthly summary reports. Please mark it clearly so that we will know it is intended for the Bulletin. (See PDR 36: 492; 37: 637) -- P. R. M.